Appendix G: Lead Benefits Analysis

Introduction

The scientific understanding of the relationship between lead and human health is rapidly expanding. This expansion is documented in numerous EPA studies on the health effects associated with lead exposure. In a pioneering study, Schwartz et al. (U.S. EPA, 1985) quantified a number of health benefits that would result from reductions in the lead content of gasoline. The work was extended by EPA's analysis of lead in drinking water (U.S. EPA, 1986a) and by an EPA-funded study of alternative lead National Ambient Air Quality Standards (U.S. EPA, 1987).

Despite this substantial research, much uncertainty remains. While the health effects of very high levels of blood lead (PbB) are quite severe (including convulsions, coma and death from lead toxicity) and have been known for many years, the effects of lower lead doses continue to be the subject of intensive scientific investigation. Dose-response functions are available for only a handful of health endpoints associated with elevated blood lead levels. Other known or strongly suspected health endpoints cannot be quantified due to a lack of information on the relationship between dose and effect. Table G-1 presents the health effects that are quantified in this analysis, as well as important known health effects that are not quantified.

Some of the health effects that are quantified in this analysis have not been estimated in previous EPA analyses. This is largely due to more recent information about the dose-response functions that makes it possible to expand the health effect coverage beyond what was done previously. Recent information is available for previously unquantified health effects, and new information on previously estimated dose-response functions is also available.

Table G-1. Quantified and Unquantified Health Effects of Lead.

Population Group	Quantified Health Effect	Unquantified Health Effect
Adult Male	*For men in specified age ranges:* Hypertension Non-fatal coronary heart disease Non-fatal Strokes Mortality	Quantified health effects for men in other age ranges Other cardiovascular diseases Neurobehavioral function
Adult Female	*For women in specified age ranges:* Non-fatal coronary heart disease Non-fatal stroke Mortality	Quantified health effects for women in other age ranges Other cardiovascular diseases Reproductive effects Neurobehavioral function
Children	IQ loss effect on lifetime earnings IQ loss effects on special educational needs Neonatal mortality due to low birth weight caused by maternal exposure to lead	Fetal effects from matemal exposure (including diminished IQ) Other neurobehavioral and physiological effects Delinquent and anti-social behavior

Methods Used to Measure and Value Health Effects

The following sections present relevant dose-response relationships for three population groups: children, men, and women. These sections also discuss data sources used for the dose-response relationships, although an extensive review of the literature is not given.[1] In addition, each section includes the methods used to value the changes in health effects determined using these dose-response relationships.

Health Benefits to Children

Changes in IQ

Elevated Pb levels may induce a number of effects on the human nervous system. Generally, these neurobehavioral effects are more serious for children than for adults because of children's rapid rate of development. It is believed that neurobehavioral deficits in children may result from both pre-natal and post-natal exposure. These nervous system effects may include hyperactivity, behavioral and attentional difficulties, delayed mental development, and motor and perceptual skill deficits. Quantification of certain manifestations of these effects is possible because sufficient data exist to estimate a dose-response relationship and IQ loss. The relationship used in the analysis is discussed below.

Quantifying the Relationship Between Blood Lead Levels and IQ

A dose-response relationship for IQ decrements has been estimated by a meta-analysis of seven research studies.[2] Regression coefficients for each study were used to determine a weighted average linear regression coefficient for the relationship between lead and IQ. Each regression coefficient was weighted by the inverse of the variance of each estimate. To determine an overall coefficient, the regression coefficients for studies that used natural logarithms of lead as the exposure index were linearized. In general, the coefficient was linearized in the blood lead range of 10 to 20 µg/dL. However, in one study (Bellinger et al., 1991), 70 percent of the data were below 10 µg/dL; thus, the Bellinger data were linearized in the 5 to 15 µg/dL range. For the studies that did not transform lead concentrations, the regression coefficients were used directly. Given the typical uncertainty within individual studies, the variation in the regression coefficients among studies was not more than would be expected. The relationship determined by Schwartz (1993) suggests that for a 1 µg/dL increase in lead, a decrease of 0.25 IQ points can be expected. The p-value (< 0.0001) indicates that this relationship is highly significant.

To obtain the total change in number of IQ points for a population of children, the 0.25 points lost per µg/dL change in blood lead is multiplied by the average blood lead level for that population. The average blood lead level modeled in this analysis is a geometric mean, not an arithmetic mean. To adjust for this, we use a relationship between the expected value and the geometric mean of a lognormally distributed random variable:

$$E[x] = \exp 1n\left[(GM) + \frac{(1n\,(GSD))^2}{2}\right] \qquad (1)$$

where E(X) is the expected value (mean) of the distribution, GM is the geometric mean, and GSD is the geometric standard deviation. Taking the natural logarithm of Equation 1 and rearranging gives the ratio between the expected value and the GM:

$$1n\,(E(X)) - 1n\,(GM) = \frac{(1n\,(GSD))^2}{2} \qquad (2)$$

$$1n\left[\frac{E(X)}{GM}\right] = \frac{(1n\,(GSD))^2}{2} \qquad (3)$$

$$\frac{E(X)}{GM} = \exp\left[\frac{(1n\,(GSD))^2}{2}\right] \qquad (4)$$

For a GSD of 1.6 (the assumed GSD of children's blood lead levels[3]), the resulting ratio between E(X) and GM is 1.117. This ratio is used in equation 5.

[1] For a detailed review of this literature see U.S. Environmental Protection Agency, (1986b) *Air Quality Criteria Document for Lead*, and 1989 Addendum. Environmental Criteria and Assessment Office, Office of Research and Development, March.

[2] Schwartz, 1993.

[3] Suggested value for sub-populations provided by IEUBK guidance manual (U.S. EPA, 1994).

The total lost IQ points for each group was estimated as:

$$(TOTAL\ LOST\ IQ)_k = \Delta GM_k \times 1.117 \times 0.25 \times (Pop)_k / 7 \quad (5)$$

where $(Pop)_k$ represents the number of children (up to age six) around a given industrial source (in the case of estimating benefits from reduced industrial emissions) or the total U.S. population of children (in the case of estimating benefits from reductions in gasoline lead emissions).

As shown in equation 5, the population of children up to age six is divided by seven to avoid double counting. If we assume that children are evenly distributed by age, this division applies this equation to only children age 0-1. If we did not divide, this equation would count a child who is age zero in the first year of the analysis and count that same child 6 more times in successive years. Dividing by seven does create some undercounting because in the first year of the analysis children from age 1 to 6 are not accounted for, while presumably they are affected by the lead exposure.

The analysis assumes a permanent loss of IQ based on blood lead levels estimated for children six years and younger. Recent studies[4] provide concrete evidence of long-term effects from childhood lead exposure.

Valuing Changes in Children's Intelligence

Available economic research provides little empirical data for society's willingness to pay (WTP) to avoid a decrease in an infant's IQ. Some research, however, has addressed monetization of *a subset* of the effects of decreased IQ. These effects would represent components of society's WTP to avoid IQ decreases. Employed alone, these monetized effects should underestimate society's WTP. Nevertheless, for the purpose of this analysis, these effects are used to approximate the WTP to avoid IQ decrements.

IQ deficits incurred through lead exposure are assumed to persist throughout the exposed infant's lifetime. Two consequences of this IQ decrement are then considered: the decreased present value of expected lifetime earnings for the infant, and the increased educational resources expended for a infant who becomes mentally handicapped or is in need of compensatory education as a consequence of lead exposure. The value of foregone earnings is addressed in this section.

The reduction in IQ has a direct and indirect effect on earnings. The direct effect is straightforward: lower IQs decrease job attainment and performance. Reduced IQ also results in reduced educational attainment, which, in turn, affects earnings and labor force participation. These effects on earnings are additive since the studies used for this analysis have controlled for these effects separately.[5] If personal decisions about the total amount of education and labor force participation were based entirely on each individual maximizing the expected present value of lifetime income, the magnitude of the indirect effect on income of a small change in educational attainment would be close to zero,[6] and certainly less than the magnitude of the direct effect. However, individuals make educational decisions based on a number of considerations in addition to the effect on the present value of lifetime earnings, such as satisfaction (utility) derived from formal education, non-compensation aspects of alternative career opportunities, the ability to pay educational costs, etc. Such considerations could lead to either a positive or negative marginal return to education. Studies[7] of educational attainment and lifetime earnings have generally identified a positive marginal return to education, suggesting that the educational attainment decision may not be based simply on expected earnings.

This analysis uses two sets of estimates of the effects of IQ on earnings. The first estimate, used by Abt Associates in a previous analysis, is based on several older studies. The second estimate is based on Salkever (1995).

Older Estimate of the Effect of IQ on Earnings: The Direct Effect of IQ on Wage Rate

Henry Aaron, Zvi Griliches, and Paul Taubman have reviewed the literature examining the relation-

[4] For example, Bellinger (1992).

[5] IQ is also correlated with other socio-economic factors which have not been quantified in this analysis.

[6] This is a straightforward result of the "envelope theorem" in economics. In this context, the envelope theorem shows that if individuals select the level of education that maximizes expected income, then the marginal benefit of additional education (i.e., the partial derivative of income with respect to education) will be zero at that optimal education level.

[7] Including Chamberlain and Griliches (1977), Ashenfelter and Ham (1979), and Salkever (1995)

ship between IQ and lifetime earnings.[8] They found that the direct effect, (schooling held constant) of IQ on wage rates ranged from 0.2 percent to 0.75 percent per IQ point. Perhaps the best of these studies is Griliches (1977).[9] He reported the direct effect of IQ on wage rates to be slightly more than 0.5 percent per IQ point. Because this is roughly the median estimate of the U.S. EPA review of the literature, this estimate is used.

Older Estimate of the Effect of IQ on Earnings: The Indirect Effect of IQ on Earnings

From Needleman et al. (1990) it is possible to estimate the change in years of schooling attained per one IQ point change. The study's regression coefficients for the effect of tooth lead on achieved grade provide an estimate of current grade achieved. However, many of these children were in college at the time and are expected to achieve a higher grade level. Following Schwartz (1990), after adjusting the published results for the fact that a higher percentage of children with low tooth lead were attending college, a 0.59 year difference in expected maximum grade achieved between the high and low exposure groups was estimated. It is assumed that educational attainment relates with blood lead levels in proportion to IQ. The difference in IQ score between the high and low exposure group was 4.5 points (from Needleman et al. (1990)). Dividing 0.59/4.5 = 0.131 suggests that the increase in lead exposure which reduces IQ by one point may also reduce years of schooling by 0.131 years.

Studies that estimate the relationship between educational attainment and wage rates (while controlling for IQ and other factors) are less common. Chamberlain and Griliches (1977) estimate that a one year increase in schooling would increase wages by 6.4 percent. In a longitudinal study of 799 subjects over 8 years, Ashenfelter and Ham (1979) reported that an extra year of education increased the average wage rate over the period by 8.8 percent. We use the average of these two estimates (7.6 percent) to calculate the indirect effect of increased schooling on the present value of lifetime income. Increased wages per IQ point are calculated using: (7.6 percent wage increase/school year) x (0.131 school years/IQ) = 1.0 percent increase in earnings per IQ point.

There is one final indirect effect on earnings. Changes in IQ affect labor force participation. Failure to graduate high school, for example, correlates with participation in the labor force, principally through higher unemployment rates and earlier retirement ages. Lead is also a strong correlate with attention span deficits, which likely reduce labor force participation. The results of Needleman et al. (1990) relating lead to failure to graduate high school can be used to estimate changes in earnings due to labor force participation. Using the odds ratio from Needleman et al., it was estimated that a one IQ point deficit would also result in a 4.5 percent increase in the risk of failing to graduate. Krupnick and Cropper (1989) provide estimates of labor force participation between high school graduates and non-graduates, controlling for age, marital status, children, race, region, and other socioeconomic status factors. Based on their data, average participation in the labor force is reduced by 10.6 percent for persons failing to graduate from high school. Because labor force participation is only one component of lifetime earnings (i.e., earnings = wage rate X years of work), this indirect effect of schooling is additive to the effect on wage rates. Combining this estimate with the Needleman result of 4.5 percent increase in the risk of failing to graduate high school per IQ point, indicates that the mean impact of one IQ point loss is a (10.6 percent x 4.5 percent) = 0.477 percent decrease in expected earnings from reduced labor force participation.

Combining the direct effect of 0.5 percent with the two indirect effects (1.0 percent for less schooling and 0.477 percent for reduced labor force participation) yields a total of 1.97 percent decrease in earnings for every loss of one IQ point.

Newer Estimate of the Effect of IQ on Earnings: Salkever (1995)

One of the most recent studies of the effects of IQ on earnings is Salkever (1995). Such an analysis with more recent data is valuable because the labor market has undergone many changes over the quarter century in which earlier studies have appeared. Like the analysis of the effect of IQ on earnings presented above, Salkever (1995) estimates this as the sum of direct and indirect effects. The *direct* effect is the sum of effects of IQ test scores on employment and earn-

[8] U.S. EPA, 1984.

[9] Griliches used a structural equations model to estimate the impact of multiple variables on an outcome of interest. This method has conceptual advantages over other empirical estimates used in the literature because it successfully controls for the many confounding variables that can affect earnings.

ings for employed persons, holding years of schooling constant. The *indirect* effect works through the effect of IQ test scores on years of schooling attained, and the subsequent effect of years of schooling on the probability of employment, and on earnings for employed persons.

Salkever (1995) provides updated estimates all of the necessary parameters using the most recent available data set, the National Longitudinal Survey of Youth (NLSY). Three regression equations provide these parameters. The years of schooling regression shows the association between IQ scores and highest grade achieved, holding background variables constant. The employment regression shows the association between IQ test scores, highest grade, and background variables on the probability of receiving earned income. This regression thus provides an estimate of the effect of IQ score on employment, holding schooling constant, and the effect of years of schooling on employment, holding IQ constant. The earnings regression shows the association between IQ test scores, highest grade, and background variables on earnings, for those with earned income.

These regressions provide parameters needed to estimate the total effect of a loss of an IQ point on earnings. The direct effects of IQ on employment and earnings for employed persons, holding schooling constant, come from the employment and earnings regressions. The indirect effect of IQ on employment through schooling is the product of the effect of IQ on years of schooling, from the years of schooling regression, and the effect of highest grade on employment, from the employment regression. The indirect effects of IQ on earnings for employed persons through schooling is the product of the effect of IQ on years of schooling, from the years of schooling regression, and the effect of highest grade on earnings for employed persons, from the earnings regression.

The total estimated effect of a loss of an IQ point on earnings is larger than the previous estimate of 1.97 percent. Based on the Salkever study, the most recent estimate of the effect of an IQ point loss is now a reduction in earnings of 1.93 percent for men and 3.22 percent for women, which is a participation-weighted average of 2.39 percent.

Value of Foregone Earnings

In the next step to monetize intelligence effects, the percent earnings loss estimate must be combined with an estimate of the present value of expected lifetime earnings. Data on earnings for employed persons and employment rates as a function of educational attainment, age, and gender were reported for the U.S. population in 1992 by the Bureau of the Census.[10] Assuming this distribution of earnings for employed persons and labor force participation rates remains constant over time, and further assuming a trend rate of real wage growth (productivity effect), an annual discount factor, and year-to-year survival probabilities, the current Census data on earnings can be used to calculate the mean present value of lifetime earnings of a person born today. This analysis assumed a person received earned income from age 18 to age 64, and assumed a real wage growth rate of one percent and an annual discount rate of five percent. Men tend to earn more than women because of higher wage rates and higher labor force participation. However, for both men and women, expected lifetime earnings increase greatly with education.

While the Census data are most likely the best available basis for projecting lifetime earnings, a number of uncertainties deserve mention. Labor force participation rates of women, the elderly, and other groups will most likely continue to change over the next decades. Real earnings of women will probably continue to rise relative to real earnings of men. Unpredictable fluctuations in the economy's growth rate will probably affect labor force participation rates and real wage growth of all groups. Medical advances will probably raise survival probabilities.

One problem that was addressed was the fact that the current educational distribution for older persons today is an especially poor predictor of educational attainment for those born today, since educational attainment has risen over time. In fact, if one simply projected educational attainment for a person born today using this method, this person would lose years of schooling with age (starting between ages 40 and 50), since average years of schooling declines with age in a one-time snapshot of the current population. To address this issue, the analysis assumes education levels cannot fall as a person ages.

Note that use of earnings is an incomplete measure of an individual's value to society. Those individuals who choose not to participate in the labor force for all of their working years must be accounted for, since the lost value of their productive services may not be accurately measured by wage rates. The largest group are those who remain at home doing housework and child rearing. Also, volunteer work contrib-

[10] U.S. Department of Commerce, 1993

utes significantly to social welfare and rates of volunteerism tend to increase with educational attainment and income.[11] If the opportunity cost of non-wage compensated work is assumed to be the average wage earned by persons of the same sex, age, and education, the average lifetime earnings estimates would be significantly higher and could be approximated by recalculating the tables using full employment rates for all age/sex/education groups. To be conservative, only the value of lost wages is considered in this analysis.

The adjusted value of expected lifetime earnings obtained above is a present value for an individual entering the labor force at age 18 and working until age 64. Given a five percent discount rate, the other assumptions mentioned, and current survival probabilities,[12] the present value of lifetime earnings of a person born today would be $170,169.

Costs of Additional Education

The increase in lifetime earnings from additional education is the gross return to education. The gross return to education, however, does not reflect the cost of the additional education. The cost of the marginal education must be subtracted from the gross return in order to obtain the net increase per IQ point from additional education. There are two components of the cost of marginal education; the direct cost of the education, and the opportunity cost of lost income during the education. An estimate of the educational cost component is obtained from the U.S. Department of Education.[13] The marginal cost of education used in this analysis is assumed to be $5,500 per year. This figure is derived from the Department of Education's reported ($5,532) average per-student annual expenditure (current plus capital expenditures) in public primary and secondary schools in 1989-'90. For comparison, the reported annual cost of college education (tuition, room and board) in 4 year public institutions is $4,975, and $12,284 for private institutions.

The estimated cost of an additional 0.131 years of education per IQ point (from the older estimate of IQ effects) is $721 (i.e., 0.131 x $5,500). Because this marginal cost occurs at the end of formal education, it must be discounted to the time the exposure and damage is modeled to occur (age zero). The average level of educational attainment in the population over age 25 is 12.9 years.[14] Therefore, the marginal educational cost is assumed to occur at age 19, resulting in a discounted present value cost of $285.

The other component of the marginal cost of education is the opportunity cost of lost income while in school. Income loss is frequently cited as a major factor in the decision to terminate education, and must be subtracted from the gross returns to education. An estimate of the loss of income is derived assuming that people in school are employed part time, but people out of school are employed full time. The opportunity cost of lost income is the difference between full-time and part-time earnings. The median annual income of people ages 18-24 employed full-time is $16,501, and $5,576 for part-time employment.[15] The lost income associated with being in school an additional 0.131 years is $1,431, which has a present discounted value at age zero of $566.

Salkever found a smaller effect of IQ on educational attainment (0.1007 years per IQ point, versus 0.131 years), which results in smaller estimated costs. Using the same method and data described above, the estimated present value of educational cost per IQ point is $219, and the income opportunity cost is $435.

Final Estimate of the Effect of IQ on Earnings.

Combining the value of lifetime earnings with the two estimates of percent wage loss per IQ point yields a low estimate of $170,169 x 1.97 percent = $3,000 per lost IQ point, and a higher estimate of $4,064 based on Salkever (1995). Subtracting the education and opportunity costs reduces these values to $2,505 and $3,410 per IQ point, respectively. This analysis uses the midpoint of these two estimates, which is $2,957. Of course, changing the discount rate would change this estimate. With an assumed discount rate of seven percent, the final estimate is only $1,311. With an assumed discount rate of three percent, the final estimate rises to $6,879.

[11] U.S. Department of Commerce, 1986. Table No. 651, p. 383.

[12] Special education costs for children who do not survive to age 18 are not counted, which results in some underestimation of benefits. However, most child mortality occurs before the age of 7, when the special education begins, so this under-counting is not substantive.

[13] "Digest of Education Statistics". U.S. Dept. of Education, 1993.

[14] "Digest of Education Statistics". U.S. Dept. of Education, 1993.

[15] "Money Income of Households, Families, and Persons in the United States: 1992". U.S. Department of Commerce, 1993.

Children with IQs Less Than 70

Quantifying the Number of Children with IQs Less than 70

In addition to the total IQ point decrements that can be predicted to occur in a population of children having a specified blood lead distribution, increases are also expected to occur in the incidence of children having very low IQ scores as the mean blood lead level for that population increases. IQ scores are normalized to have a mean of 100 and a standard deviation of fifteen. An IQ score of 70, which is two standard deviations below the mean, is generally regarded as the point below which children require special compensatory education tailored to the mentally handicapped.

The relationship presented here for estimating changes in the incidence of IQ < 70 was developed to make use of the most current IQ point decrement function provided by Schwartz (1993). It is assumed that for a baseline set of conditions where a population of children has a blood lead distribution defined by some geometric mean and geometric standard deviation, that population also has a normalized IQ point distribution with a mean of 100 and a standard deviation of 15. For this baseline condition, the proportion of the population expected to have IQ < 70 is determined from the standard normal distribution function:

$$P(IQ<70) = \Phi(z) \qquad (6)$$

where:

P(IQ <70) = Probability of IQ scores less than 70

z = standard normal variate; computed for an IQ score of 70, with mean IQ score of 100 and standard deviation of 15 as:

$$z = \frac{70 - 100}{15} = -2 \qquad (7)$$

$\Phi(z)$ = Standard normal distribution function:

$$\frac{1}{\sqrt{2\pi}} \int_{-\infty}^{z} e^{-\frac{u^2}{2}} \, du \qquad (8)$$

The integral in the standard normal distribution function does not have a closed form solution. Therefore, values for $\Phi(z)$ are usually obtained readily from software with basic statistical functions or from tables typically provided in statistics texts. The solution for $\Phi(z)$ where z = -2 is 0.02275. That is, for the normalized IQ score distribution with mean of 100 and standard deviation of 15, it is expected that approximately 2.3 percent of children will have IQ scores below 70.

To estimate changes in the proportion of children with IQ scores below 70 associated with changes in mean blood lead levels for a population of children, the following two key assumptions are made:

1. The mean IQ score will change as a result of changes in the mean blood lead level as:
   $$\Delta\overline{IQ} = -0.25 \times \Delta\overline{PbB}$$
   where
   $$\Delta\overline{IQ} \text{ and } \Delta\overline{PbB}$$
   are the changes in the mean IQ score and in the mean blood lead levels, respectively, between the no-control and control scenarios. This relationship relies on Schwartz' estimate (1993) of a decrease of 0.25 IQ points for each µg/dL increase in blood lead. Note that the mean blood lead level referred to here is the arithmetic mean (or expected value) for the distribution obtained as described previously from the GM and GSD.

2. The standard deviation for the IQ distribution remains at 15.

Using these assumptions, the change in the proportion of children having IQ <70 can then be determined for a given change in mean blood lead from:

$$\Delta P(IQ<70) = \Phi(z_{No\text{-}control}) - \Phi(z_{control}) = \Phi(z_{No\text{-}control}) - 0.02275 \qquad (9)$$

where,

$$z_{No\text{-}control} = \frac{70 - (100 + 0.25 \times \Delta\overline{PbB})}{15} \qquad (10)$$

For a given change in PbB between the control and no-control scenarios a response in terms of IQ is calculated. The procedure above yields an estimate of the percent of the population with IQs less than 70. This percentile is multiplied by the exposed population of children to estimate the increased incidence of

children with low IQs. As in the IQ point loss equation, the results of this function are applied to children age 0-6 and divided by seven to avoid double counting. (See discussion under equation 5).

This procedure quantifies only the change in the number of children who pass below the IQ=70 threshold. Any other changes in children's IQ are quantified using the IQ point loss function described previously. Treating these two endpoints additively does not result in double counting, because the value associated with the IQ point loss function is the change in worker productivity while the value associated with IQs less than 70 is the increased educational costs for the individual, as discussed below.

Valuing the Reduction in Number of Children with IQs less than 70

To value the reduction in the number of children with IQs less than 70, the reduction in education costs were measured - a clear underestimate of the total benefits.[16] Kakalik et al. (1981), using data from a study prepared for the Department of Education's Office of Special Education Programs, estimated that part-time special education costs for children who remained in regular classrooms cost $3,064 extra per child per year in 1978. Adjusting for inflation and real income growth using the GNP price deflator yields an estimate of $6,318 per child in 1990 dollars. For the calculations, this incremental estimate of the cost of part-time special education was used to estimate the cost per year per child needing special education as a result of impacts of lead on mental development. Costs would be incurred from grades one through twelve. Discounting future expenses at a rate of three percent yields an expected present value cost of approximately $52,700 per infant (assuming compensatory education begins at age 7 and continues through age 18). Note that this underestimates the cost, since Kakalik et al. measured the increased cost to educate children attending regular school — not a special education program.

Changes in Neonatal Mortality

Quantifying the relationship between PbB levels and neonatal mortality

U.S. EPA (1990c) cites a number of studies linking fetal exposure to lead (via *in utero* exposure from maternal intake of lead) to several adverse health effects. These effects include decreased gestational age, reduced birth weight, late fetal death, and increases in infant mortality.[17] The Centers for Disease Control (CDC, 1991a) presents a method to estimate changes in infant mortality due to changes in maternal blood lead levels during pregnancy.[18] The analysis links two relationships. The first relationship, between maternal blood lead level and gestational age of the newborn, was estimated by Dietrich et al. (1987). CDC then estimated infant mortality as a function of gestational age, using data from the Linked Birth and Infant Death Record Project from the National Center for Health Statistics. The resulting association is a decreased risk of infant mortality of 10^{-4} (or 0.0001) for each 1 µg/dL decrease in maternal blood lead level during pregnancy. This is the relationship used in the current analysis.

Valuing changes in neonatal mortality

The central estimate of the monetary benefit associated with reducing risks of neonatal mortality is $4.8 million per avoided mortality. This analysis attempts to capture the credible range of uncertainty associated with this estimate by describing the monetary benefit as a distribution of values: a Weibull distribution with a mean value of $4.8 million and a standard deviation of $3.24 million. Appendix I documents the derivation of this distribution and the sources of uncertainty in valuing reduced mortality risks.

Health Benefits to Men

In addition to adversely affecting children's health, lead exposure has also been shown to adversely affect adults. The health effects in adults that are quantified and included in the benefits analysis are all re-

[16] The largest part of this benefit is the parents' willingness to pay to avoid having their child become mentally handicapped, above and beyond the increased educational costs.

[17] Due to unavailability of suitable data, non-fatal health impacts due to decreased gestational age or reduced birth weight have not been included in this analysis. For example, the benefits from avoided developmental disabilities such as sensory and motor dysfunction associated with decreased gestational age have not been included.

[18] The estimated change in infant mortality due to change in birth weight was not modeled because the data relating prenatal lead exposure to birth weight are not as strong as data relating lead exposure and gestational age.

lated to the effects of lead on blood pressure.[19] The estimated relationships between these health effects and lead exposure differ between men and women. The quantified health effects include increased incidence of hypertension (estimated for males only), initial coronary heart disease (CHD), strokes (initial cerebrovascular accidents and atherothrombotic brain infarctions), and premature mortality. Other health effects associated with elevated blood pressure, and other adult health effects of lead including neurobehavioral effects, are not included in this analysis. This section describes the quantified health effects for men; the next section describes the health effects for women.

Hypertension

Quantifying the relationship between PbB levels and hypertension

Elevated blood lead has been linked to elevated blood pressure (BP) in adult males, especially men aged 40-59 years.[20] Further studies have demonstrated a dose-response relationship for hypertension (defined as diastolic blood pressure above 90 mm Hg for this model) in males aged 20-74 years.[21] This relationship is:

$$\Delta Pr(HYP) = \frac{1}{1 + e^{2.744 - .793*(\ln PbB_1)}} - \frac{1}{1 + e^{2.744 - .793*(\ln PbB_2)}} \quad (11)$$

where:

ΔPr(HYP) = the change in the probability of hypertension;

PbB_1 = blood lead level in the control scenario; and

PbB_2 = blood lead level in the no-control scenario.

Valuing reductions in hypertension

The best measure of the social costs of hypertension, society's willingness to pay to avoid the condition, cannot be quantified without basic research well beyond the scope of this project. Ideally, the measure would include all the medical costs associated with treating hypertension, the individual's willingness to pay to avoid the worry that hypertension could lead to a stroke or CHD, and the individual's willingness to pay to avoid changes in behavior that may be required to reduce the probability that hypertension leads to a stroke or CHD. Medical costs of hypertension can be divided into four categories: physician charges, medication costs, hospitalization costs and lost work time.

This analysis uses recent research results to quantify two components of this benefit category. Krupnick and Cropper (1989), using data from the National Medical Care Expenditure Survey, have estimated the medical costs of hypertension. These costs include physician care, drugs and hospitalization costs. In addition, hypertensives have more bed disability days and work loss days than others of their age and sex. Krupnick and Cropper estimated the increase in work loss days at 0.8 per year, and these were valued at the mean daily wage rate. Adjusting the above costs to 1990 dollars gives an estimate of the annual cost of each case of hypertension of $681. The estimate is likely to be an underestimate of the true social benefit of avoiding a case of hypertension for several reasons. First, a measure of the value of pain, suffering and stress associated with hypertension is not included. Second, the direct costs (out-of-pocket expenses) of diet and behavior modification (e.g., salt-free diets, etc.) are not valued. These costs are likely to be significant, since modifications are typically severe. Third, the loss of satisfaction associated with the diet and behavior modifications are ignored. Finally, the medication for hypertension may produce side effects including drowsiness, nausea, vomiting, anemia, impotence, cancer, and depression. The benefits of avoiding these side effects are not included in this estimate.

Quantifying the relationship between blood lead and blood pressure

Because blood lead has been identified as a risk factor in a number of cardiovascular illnesses,[22] it is useful to quantify the effect of changes in blood lead levels on changes in blood pressure for reasons other than predicting the probability of hypertension. Based on results of a meta-analysis of several studies, Schwartz (1992a) estimated a relationship between a

[19] Citing laboratory studies with rodents, U.S. EPA (1990c) also presents evidence of the genotoxicity and/or carcinogenicity of lead compounds. While such animal toxicological evidence suggests that human cancer effects are possible, dose-response relationships are not currently available.

[20] Pirkle et al., 1985.

[21] Schwartz, 1988.

[22] Shurtleff, 1974; McGee and Gordon, 1976; Pooling Project Research Group, 1978.

change in blood pressure associated with a decrease in blood lead from 10 µg/dL to 5 µg/dL.[23] The coefficient reported by Schwartz leads to the following function relating blood pressure to blood lead for men:

$$\Delta DBP_{men} = 1.4 \times 1n\left(\frac{PbB_1}{PbB_2}\right) \qquad (12)$$

where:

ΔDBP_{men} = the change in men's diastolic blood pressure expected from a change in PbB;
PbB_1 = blood lead level in the control scenario (in µg/dL); and
PbB_2 = blood lead level in the no-control scenario (in µg/dL).

This blood lead to blood pressure relationship is used to estimate the incidence of initial coronary heart disease, strokes (atherothrombotic brain infarctions and initial cerebrovascular accidents) and premature mortality in men.

Changes In Coronary Heart Disease

Quantifying the relationship between blood pressure and coronary heart disease

Estimated blood pressure changes can be used to predict the increased probability of the initial occurrence of CHD and stroke.[24] Increased blood pressure would also increase the probability of reoccurrence of CHD and stroke, but these quantified relationships are not available. First-time coronary heart disease events in men can be predicted using an equation with different coefficients for each of three age groups. For men between 40 and 59 years old, information from a 1978 study by the Pooling Project Research Group (PPRG) is used. PPRG (1978) presents a multivariate model (controlling for smoking and serum cholesterol) that relates the probability of coronary heart disease (CHD) to blood pressure. The model used data from five different epidemiological studies. From this study, the equation for the change in 10-year probability of occurrence of CHD is:

$$\Delta Pr(CHD_{40\text{-}59}) = \frac{1}{1 + e^{4.996 - 0.030365 * DBP_1)}} - \frac{1}{1 + e^{4.996 - 0.030365 * DBP_2)}} \qquad (13)$$

where:

$\Delta Pr(CHD_{40\text{-}59})$ = change in 10-year probability of occurrence of CHD event for men between 40-59 years old,
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

The relationship between BP and first-time CHD in older men was determined from information presented in Shurtleff (1974). This study also uses data from the Framingham Study (McGee and Gordon, 1976) to estimate univariate relationships between BP and a variety of health effects by sex and for each of the following age ranges: 45-54, 55-64, and 65-74 years. Single composite analyses for ages 45-74 were also performed for each sex. For every equation, t-statistics on the variable blood pressure are significant at the 99th percent confidence interval. For men aged 60 to 64 years old, first-time CHD can be predicted from the following equation:

$$\Delta Pr(CHD_{60\text{-}64}) = \frac{1}{1 + e^{5.19676 - 0.02351 * DBP_1)}} - \frac{1}{1 + e^{5.19676 - 0.02351 * DBP_2)}} \qquad (14)$$

where:

$\Delta Pr(CHD_{60\text{-}64})$ = change in 2 year probability of occurrence of CHD event for men from 60 to 64 years old;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

For men aged 65 to 74 years old, the following equation uses data from Shurtleff (1974) to predict the probability of first-time CHD:

$$\Delta Pr(CHD_{65\text{-}74}) = \frac{1}{1 + e^{4.90723 - 0.02031 * DBP_1)}} - \frac{1}{1 + e^{4.90723 - 0.02031 * DBP_2)}} \qquad (15)$$

where:

$\Delta Pr(CHD_{65\text{-}74})$ = change in 2 year probability of occurrence of CHD event for men from 65 to 74 years old;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

[23] Schwartz, 1992a.

[24] U.S. EPA, 1987.

The probability changes calculated using the functions above are used to estimate the number of CHD events avoided in a given year due to air quality improvements attributable to the Clean Air Act. The resulting CHD incidence estimates include both fatal and non-fatal events. However, because mortality benefits are independently estimated in this analysis, it is important to capture only the non-fatal CHD events. Shurtleff (1974) reported that two-thirds of all CHD events were non-fatal. This factor was therefore applied to the estimate of avoided CHD events for each age category.

Valuing reductions in CHD events

General methodology

Because of the lack of information on WTP to avoid an initial CHD event, WTP was estimated by estimating the associated cost of illness (COI). This will underestimate WTP, as explained in Appendix I. Full COI consists of the present discounted value of all costs associated with the event, including both direct and indirect costs incurred during the hospital stay, as well as the present discounted values of the streams of medical expenditures (direct costs) and lost earnings (indirect costs) incurred once the individual leaves the hospital.

Wittels et al. (1990) estimate the total medical costs within 5 years of diagnosis of each of several types of CHD events (including acute myocardial infarction, angina pectoris, unstable angina pectoris, sudden death and nonsudden death) examined in the Framingham Study. Costs were estimated by multiplying the probability of a medical test or treatment within five years of the initial CHD event (and associated with that event) by the estimated price of the test or treatment. All prices were in 1986 dollars. (It does not appear that any discounting was used.) The probabilities of tests or treatments were based on events examined in the Framingham Study. The authors estimate a total expected cost over a five year period (in 1986 dollars) of $51,211 for acute myocardial infarction, $24,980 for angina pectoris, and $40,581 for unstable angina pectoris. Converted to 1990 dollars (using the consumer price index for medical care, U.S. Bureau of the Census, 1992), this is $68,337 for acute myocardial infarction, $33,334 for angina pectoris, and $54,152 for unstable angina pectoris. (The figures for sudden death and nonsudden death are not included because the CHD events in this analysis exclude those that resulted in death, to avoid double counting.)

Cropper and Krupnick (1990) suggest, in an unpublished study, that CHD-related lost earnings could be a significant component of total COI, although the value of earnings lost may vary substantially with the age of onset of CHD. They estimate, for example, that an individual whose first heart attack occurs between ages 55 and 65 will have an expected annual earnings loss of $12,388 (in 1990 dollars), and a present discounted value of lost earnings over a five-year period of about $53,600, using a five percent discount rate. This is almost as much as the total medical costs over 5 years estimated by Wittels et al. (1990) for unstable angina pectoris, and substantially more than the corresponding estimate of medical costs for angina pectoris. For an individual whose first heart attack occurs between ages 45 and 54, on the other hand, Cropper and Krupnick estimate annual average lost earnings of $2,143 (in 1990 dollars), and a present discounted value of lost earnings over a five-year period of about $9,300, again using a five percent discount rate. Cropper and Krupnick do not estimate medical costs for exactly the same disease categories as Wittels et al., but their research suggests that whether the five-year COI of a CHD event, including both medical costs and lost earnings, is lower or higher than the average of the three estimates reported by Wittels et al. depends on an individual's age at the onset of CHD. Combining Cropper and Krupnick's five-year lost earnings estimates with their estimates for average annual medical expenditures for ischemic heart disease summed over five years, for example, yields a total COI of about $47,000 for a 50 year old and $72,000 for a 60 year old, compared to the $52,000 average of the three estimates reported by Wittels et al.

In addition to the variability in estimates of medical costs and lost earnings arising from CHD, there is uncertainty regarding the proportion of pollution-related CHD events associated with the various classes of CHD. To characterize this uncertainty it was assumed, in the absence of further information, that all pollution-related CHD events are either acute myocardial infarctions, angina pectoris, or unstable angina pectoris. A distribution of estimates of COI for pollution-related CHD was generated by Monte Carlo methods. On each iteration, a value was randomly selected from each of three continuous uniform distributions. Each value selected was normalized by

dividing by the sum of the three values, so that the three normalized values summed to 1.0. The resulting triplet of proportions represents a possible set of proportions of pollution-related CHD events that are acute myocardial infarction, angina pectoris, and unstable angina pectoris. The corresponding dollar value for the iteration is a weighted average of the estimated dollar values for the three types of CHD event (from Wittels et al.), where the weights are the three randomly selected proportions. The central tendency estimate of the COI associated with a case of pollution-related CHD is the mean of this distribution, about $52,000.

This estimate is likely to understate full COI because it does not include lost earnings. It is likely to underestimate total WTP to an even greater extent because it does not include WTP to avoid the pain and suffering associated with the CHD event. It is, however, substantially greater than an estimate based only on the direct and indirect costs incurred during the hospital stay.

The valuation for CHD is additive with the valuation for hypertension despite the fact that the conditions often occur together, because the two values represent different costs associated with the conditions. The valuation for hypertension is based on loss of work days as a result of hypertension and some of the medical costs associated with treating hypertension. The valuation for CHD is based on the willingness to pay to avoid the pain and suffering of the CHD itself. Therefore, these two valuations can be separated and added together.

Changes in Initial Cerebrovascular Accidents and Initial Atherothrombotic Brain Infarctions

Quantifying the relationship between blood pressure and first-time stroke

Two types of health events are categorized as strokes: initial cerebrovascular accidents (CA) and initial atherothrombotic brain infarctions (BI). The risk has been quantified for the male population between 45 and 74 years old.[25] For initial cerebrovascular accidents, the logistic equation is:

$$\Delta Pr(CA_{men}) = \frac{1}{1 + e^{8.58889 - 0.04066 * DBP_1)}} - \frac{1}{1 + e^{8.58889 - 0.04066 * DBP_2)}} \quad (16)$$

where:

$\Delta Pr(CA_{men})$ = change in 2 year probability of cerebrovascular accident in men;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

For initial atherothrombotic brain infarctions, the logistic equation is:

$$\Delta Pr(BI_{men}) = \frac{1}{1 + e^{9.9516 - 0.04840 * DBP_1)}} - \frac{1}{1 + e^{9.9516 - 0.04840 * DBP_2)}} \quad (17)$$

where:

$\Delta Pr(BI_{men})$ = change in 2 year probability of brain infarction in men;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

Similar to CHD events, this analysis estimates only non-fatal strokes (to avoid double-counting with premature mortality). Shurtleff (1974) reported that 70 percent of strokes were non-fatal. This factor was applied to the estimates of both CA and BI.

Valuing reductions in strokes

Taylor et al. (1996) estimate the lifetime cost of stroke, including the present discounted value (in 1990 dollars) of the stream of medical expenditures and the present discounted value of the stream of lost earnings, using a five percent discount rate. Estimates are given for each of three separate categories of stroke, separately for males and females at ages 25, 45, 65, and 85. For all three types of stroke, the indirect costs (lost earnings) substantially exceed the direct costs at the two younger ages, and are about the same as or smaller than direct costs at the older ages.

Both types of stroke considered in this analysis fall within the third category, ischemic stroke, considered by Taylor et al. To derive a dollar value of avoiding an initial ischemic stroke for males, a dollar value for avoiding ischemic stroke among males age 55 was interpolated from the values for males ages 45 and 65; similarly, a dollar value for avoiding ischemic stroke among males age 75 was interpolated from the values for males ages 65 and 85. Of males in the United

[25] Shurtleff, 1974.

States between the ages of 45 and 74 (the age group for which lead-related stroke is predicted), 41.2 percent are ages 45-54 and the remaining 58.8 percent are ages 55-74. The value of an avoided stroke among males was calculated as the weighted average of the values for males in age group 45-54 and males in age group 55-74 , where the weights are the above percents. The value for age group 45-54 is the average of the values for ages 45 and 55; the value for age group 55-74 is the average of the values for ages 55, 65 and 75. The resulting average value of an avoided stroke among males aged 45-74 is about $200,000.

Changes in Premature Mortality

Quantifying the relationship between blood pressure and premature mortality

Information also exists to predict the increased probability of premature death from all causes as a function of elevated blood pressure. U.S. EPA (1987) used population mean values for serum cholesterol and smoking to reduce results from a 12 year follow-up of men aged 40-54 in the Framingham Study (McGee and Gordon, 1976) to an equation in one explanatory variable:

$$\Delta Pr(MORT_{40\text{-}54}) = \frac{1}{1 + e^{5.3158 - 0.03516 * DBP_1)}} - \frac{1}{1 + e^{5.3158 - 0.03516 * DBP_2)}} \quad (18)$$

where:

$\Delta Pr(MORT_{40\text{-}54})$ = the change in 12 year probability of death for men aged 40-54;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Information from Shurtleff (1974) can be used to estimate the probability of premature death in men older than 54 years old. This study has a 2 year follow up period, so a 2 year probability is estimated. For men aged 55 to 64 years old, mortality can be predicted by the following equation:

$$\Delta Pr(MORT_{55\text{-}64}) = \frac{1}{1 + e^{4.89528 - 0.01866 * DBP_1)}} - \frac{1}{1 + e^{4.89528 - 0.01866 * DBP_2)}} \quad (19)$$

where:

$\Delta Pr(MORT_{55\text{-}64})$ = the change in 2 year probability of death in men aged 55-64;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

For men aged 65 to 74 years old, premature mortality can be predicted by the following equation:

$$\Delta Pr(MORT_{65\text{-}74}) = \frac{1}{1 + e^{3.05723 - 0.00547 * DBP_1)}} - \frac{1}{1 + e^{3.05723 - 0.00547 * DBP_2)}} \quad (20)$$

where:

$\Delta Pr(MORT_{65\text{-}74})$ = the change in 2 year probability of death in men aged 55-64;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Valuing reductions in premature mortality

As discussed above, premature mortality is valued at $4.8 million per case (discussed further in Appendix I). Because this valuation is based on the willingness to pay to the risk of death, and the CHD valuation is based on the willingness to pay to avoid the pain and suffering of a CHD event (defined as a CHD event that does not end in death, to avoid double counting), these two endpoints are additive as well.

Health Benefits to Women

Available evidence suggests the possibility of health benefits from reducing women's exposure to lead. Recent expanded analysis of data from the second National Health and Nutrition Examination Survey[26] (NHANES II) by Schwartz (1990) indicates a significant association between blood pressure and blood lead in women. Another study, by Rabinowitz et al. (1987), found a small but demonstrable association between maternal blood lead and pregnancy hypertension and blood pressure at time of delivery.

[26] The Second National Health and Nutrition Examination Survey (NHANES II) was conducted by the U.S. Department of Health and Human Services from 1976 to 1980 and provides researchers with a comprehensive set of nutritional, demographic and health data for the U.S. population.

The effect of lead exposure on the blood pressure of women, relative to the effect on men, is examined in a review of ten published studies.[27] All of the reviewed studies included data for men, and some included data for women. A concordance procedure was used to combine data from each study to predict the decrease in diastolic BP associated with a decrease from 10 µg/dL to 5 µg/dL PbB. The results suggest that the effect on blood pressure for women of this decrease in blood lead is 60 percent of the effect of the same change observed in men. Thus, for women, Equation can be rewritten as:

$$\Delta DBP_{women} = (0.6 \times 1.4) \times 1n\left(\frac{PbB_1}{PbB_2}\right) \quad (21)$$

where:

ΔDBP_{women} = the change in women's diastolic blood pressure expected from a change in PbB;
PbB_1 = blood lead level in the control scenario; and
PbB_2 = blood lead level in the no-control scenario.

Although women are at risk of having lead-induced hypertension, there is not a dose-response function for hypertension in women available at this time. Omitting the hypertension benefits for women creates an underestimate of the total benefits, but the impact on the total benefits estimation will likely be small. Lead raises blood pressure in women less than in men, so the probability of causing hypertension is likely to be less than in men, and the total value of hypertension in men is a small portion of the overall estimated benefits.

Changes in Coronary Heart Disease

Quantifying the relationship between blood pressure and coronary heart disease

Elevated blood pressure in women results in the same effects as for men (the occurrence of CHD, two types of stroke, and premature death). However, the general relationships between BP and these health effects are not identical to the dose-response functions estimated for men. All relationships presented here have been estimated for women aged 45 to 74 years old using information from Shurtleff (1974). First-time CHD in women can be estimated from the following equation:

$$\Delta Pr(CHD_{women}) = \frac{1}{1 + e^{6.9401 - 0.03072*DBP_1)}} - \frac{1}{1 + e^{6.9401 - 0.03072*DBP_2)}} \quad (22)$$

where:

$\Delta Pr(CHD_{women})$ = change in 2 year probability of occurrence of CHD event for women aged 45-74;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

Again, non-fatal CHD events were estimated by assuming that two-thirds of the estimated events were not fatal (Shurtleff, 1974).

Valuing reductions in CHD events

Values of reducing CHD events for women are assumed to be equal to those calculated for men (above): $52,000 per CHD event.

Changes in Atherothrombotic Brain Infarctions and Initial Cerebrovascular Accidents

Quantifying the relationship between blood pressure and first-time stroke

For initial atherothrombotic brain infarctions in women, the logistic equation is:

$$\Delta Pr(BI_{women}) = \frac{1}{1 + e^{10.6716 - 0.0544*DBP_1)}} - \frac{1}{1 + e^{10.6716 - 0.0544*DBP_2)}} \quad (23)$$

where:

$\Delta Pr(BI_{women})$ = change in 2 year probability of brain infarction in women aged 45-74;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

The relationship between BP and initial cerebrovascular accidents can be predicted by the following logistic equation:

$$\Delta Pr(CA_{women}) = \frac{1}{1 + e^{9.07737 - 0.04287*DBP_1)}} - \frac{1}{1 + e^{9.07737 - 0.04287*DBP_2)}} \quad (24)$$

[27] Schwartz, 1992b.

where:

$\Delta Pr(CA_{women})$ = change in 2 year probability of cerebrovascular accident in women aged 45-74;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

The predicted incidences of avoided BI and CA were multiplied by 70 percent to estimate only non-fatal strokes (Shurtleff, 1974).

Valuing reductions in strokes

The value of avoiding an initial cerebrovascular accident or an initial atherothrombotic brain infarction for women was calculated in the same way as for men (see above). Of women in the United States between the ages of 45 and 74 (the age group for which lead-related stroke was predicted), 38.2 percent are ages 45-54 and the remaining 61.8 percent are ages 55-74. Using these percentages, and the gender- and age-specific values in Taylor et al. (1996) the average value among women ages 45-74 of avoiding either type of stroke was estimated to be about $150,000.

Changes in Premature Mortality

Quantifying the relationship between blood pressure and premature mortality

The risk of premature mortality in women can be estimated by the following equation:

$$\Delta Pr(MORT_{women}) = \frac{1}{1 + e^{5.40374 - 0.01511 * DBP_1)}} - \frac{1}{1 + e^{5.40374 - 0.01511 * DBP_2)}} \quad (25)$$

where:

$\Delta Pr(MORT_{women})$ = the change in 2 year probability of death for women aged 45-74;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Valuing reductions in premature mortality

The value of reducing premature mortality for women is assumed to be equal to that estimated for all premature mortality, $4.8 million per incident (see Appendix I).

Quantifying Uncertainty

Characterizing Uncertainty Surrounding the Dose-Response Relationships

The dose-response functions described for each health endpoint considered above generally quantify the adverse health effects expected due to increased exposure to lead. For children, these relationships are described directly in terms of changes in blood lead. For adults, effects are estimated in terms of changes in blood pressure (which are related to changes in blood lead levels). As with any estimate, uncertainty is associated with the dose-response relationships.

Consistent with the approach outlined in Appendix D for the non-lead criteria air pollutants, this analysis attempts to capture the uncertainty associated with these relationships. This is accomplished by estimating a distribution associated with each dose-response coefficient using the information reported in the literature. This analysis assumes these distributions to be normal. For each of the coefficients used to relate adverse health effects to lead exposure, Table G-2 summarizes the means and standard deviations of the normal distributions used in this analysis.

Characterizing Uncertainty Surrounding the Valuation Estimates

The procedure for quantifying uncertainty associated with the valuation estimates is similar to that used to characterize the dose-response coefficient estimates. The valuation distributions for health effects considered in the lead analysis are documented in Appendix I.

Table G-2. Uncertainty Analysis: Distributions Associated With Dose-Response Coefficients Used to Estimate Lead Health Effects.

Health Effect	Parameters of Normal distributions describing Dose-Response Coefficients	
	Mean	Standard Deviation
Blood Lead-Blood Pressure Coefficient (Adults)	1.44	0.85
Adult Males		
Mortality (ages 40-54)	0.03516	0.16596
Mortality (ages 55-64)	0.01866	0.00533
Mortality (ages 65-74)	0.00547	0.00667
Chronic Heart Disease (ages 40-59)	0.030365	0.003586
Chronic Heart Disease (ages 60-64)	0.02351	0.028
Chronic Heart Disease (ages 65-74)	0.02031	0.00901
Cerebrovascular Accidents	0.04066	0.00711
Atherothrombotic Brain Infarctions	0.0484	0.00938
Hypertension	0.793	not available
Adult Females		
Mortality (ages 45-74)	0.01511	0.00419
Chronic Heart Disease	0.03072	0.00385
Cerebrovascular Accidents	0.04287	0.00637
Atherothrombotic Brain Infarctions	0.0544	0.00754
Children		
Infant Mortality	0.0001	not available
Lost IQ Points	0.245	0.039
IQ<70 (cases)	relies on Lost IQ Point distribution	

Industrial Processes and Boilers and Electric Utilities

This section describes the methods and data sources used to estimate changes in blood lead levels due to changes in lead emissions from industrial processes and boilers between 1970 and 1990 and from electric utilities between 1975 and 1990. The estimates of the changes in health effects resulting from changes in lead emissions due to the CAA are also presented.

Methods Used to Determine Changes in Lead Emissions from Industrial Processes from 1970 to 1990

This analysis used several sources to determine the changes in facility-specific emissions of lead from industrial processes. To summarize, the analysis extracted 1990 facility-specific lead emissions data from the Toxics Release Inventory (TRI), which provides recent emissions data for over 20,000 U.S. manufacturing facilities. This study then adjusted these data by the relative changes in lead emissions between 1970 and 1990; these relative changes were derived from several data sources described below. This method yielded facility-specific emissions for five year intervals between 1970 and 1990 for both the controlled and uncontrolled scenarios. The five-year values were interpolated to derive annual changes for each year between 1970 and 1990. Specific details on this approach are given below.

TRI Data

The Toxics Release Inventory (TRI) is mandated by the Superfund Amendment Reauthorization Act (SARA) Title III Section 313 and requires that U.S. manufacturing facilities with more than 10 employees file annual reports documenting multimedia environmental releases and off-site transfers for over 300 chemicals. Facilities report both stack and fugitive releases to air. Reported releases are generally estimates rather than precise quantifications. Emissions data can be presented as numerical point estimates, or, if releases are below 1,000 pounds, as an estimated range of emissions.

From the TRI data base, this analysis extracted data from the reporting year 1990 for all facilities reporting emissions of lead to air, as either stack or fugitive emissions. Data were reported as annual emissions (in pounds per year). Where emissions are reported as a range, this analysis used the upper bound of the range to represent the emissions.[28] TRI facilities also report their location by latitude and longitude. In order to later match facilities emitting lead with Census data on surrounding exposed populations, this analysis uses the latitudes and longitudes of lead-emitting facilities.

Derivation of Industrial Process Emissions Differentials 1970-1990

The TRI database is the Agency's single best source of consistently reported release data; however, the database does not include information for most of the years modeled in this analysis. Furthermore, this analysis required estimates of hypothetical emissions in the absence of the CAA. Therefore, estimates were created for the emissions of lead from industrial sources under the CAA, and in the absence of the CAA, for the years 1970, 1975, 1980, 1985, and 1990. The *percent changes*, or differentials, reflected by these estimates were then applied to the 1990 TRI data to obtain facility-level release estimates for the years of interest for the control and no-control scenarios.

The method for creating these differentials captured the two potential causes of the differences between emissions from industrial sources regulated by the CAA and emissions from those same sources in the absence of the CAA. The first cause of the difference in emissions is a change in overall industrial output, resulting from the macroeconomic impact of the CAA. The second element is a change in emissions per unit of output, which results from the adoption of cleaner processes and the application of emissions control technology mandated by the CAA. The methods used to project the effects of these two causes, described below, were designed to be as consistent as possible with other emissions projection methods for other segments of the CAA retrospective analysis.

Data sources

Data for the differentials estimates were taken from the following sources:

- the Jorgenson/Wilcoxen (J/W) model projections, conducted as part of the section 812 analysis. This data source addresses the first cause of changes in emissions: the macroeconomic changes that resulted from the implementation of the 1970 CAA. The J/W model calculated the change in economic output for each of thirty-five industrial sectors, roughly analogous to two-digit standard industrial classification (SIC) codes, that resulted from the CAA's implementation. The specific output used from the J/W model in this analysis was the percentage change in economic output for the various industrial sectors, rather than any absolute measure of economic activity.

- the 1991 OAQPS Trends database. This data base is an emissions projection system that was used to produce the report, "The National Air Pollutant Emission Estimates, 1940-1990." It contains information on economic activity, national level emissions and emission controls, by industrial process, from 1970 through 1990. Three different elements were extracted from the Trends database: the emissions of lead per unit economic output for various industrial processes for the years 1970-1990; annual economic output data for these industrial processes; and the emission calculation formula.

- the National Energy Accounts (NEA), compiled by the Bureau of Economic Analysis. This database records the historical levels of industrial energy consumption, disaggregated by fuel type at the approximately three-digit SIC code level.

The manner in which these data were combined to derive lead emissions estimates is described below.

Estimates of industrial process emissions in the control scenario

Emissions data for industrial processes were estimated for the years 1970, 1975, 1980, 1985, and 1990. For each of these years, this analysis extracted an emission factor and a control efficiency for each lead-

[28] Ranges are infrequently reported and are either reported as 0-500 lbs. or 500-1000 lbs. The infrequency of the incidence of a facility reporting a range and the relatively small quantities of lead released by those facilities means any overestimation of benefits that results from using the upper limit of the range is extremely minor.

emitting industrial process in the Trends database. Emissions factors are expressed as amount of lead emitted per unit of economic activity, and control efficiencies are reported as the percent that emissions are reduced through the application of pollution control technology to the process. The year-specific emission factors and control efficiencies were multiplied by the economic activity data for that year, for that process, as reported in the Trends database, using the following equation found in the Trends report:

$$Emissions = (Economic\ Activity) \times (Emission\ Factor) \times (1 - Control\ Efficiency) \quad (26)$$

This calculation yielded the estimated control scenario emissions, by industrial process. Industrial processes were then assigned to an NEA code. Finally, all processes assigned to a given NEA code were summed to give a total emissions estimate for that NEA code.

Estimates of industrial process emissions in the no-control scenario

The results from the J/W model were used to estimate process emissions in the no-control scenario. As stated above, the J/W model provides percent changes in economic outputs by industrial sector. To use these values, lead-emitting industrial processes (in the Trends database) were assigned to a J/W sector. The percent change for that sector from the J/W model was then used to adjust the economic activity data for that process from the Trends database. These adjusted economic output figures were used together with 1970 emission factors and control efficiencies to derive the estimated lead emissions for each industrial process in the no-control scenario. The 1970 emission factors and control efficiencies were used for all years in the analysis (1970, 1975, 1980, 1985 and 1990) in the no-control scenario; this assumes that emissions per unit economic output and control efficiencies would have been constant over time in the absence of the CAA. This is the same approach that was used to project the changes in emissions from industrial processes for other criteria pollutants in other portions of the CAA retrospective analysis. The process-level emissions were then aggregated to the NEA-code level, as in the controlled scenario.

Matching TRI Data to Industrial Process Emissions Differentials

The methods described in the preceding section yielded emissions estimates from industrial processes in the control and no-control scenarios, by NEA code. We used these estimates to derive percent changes in emissions between control and no-control scenarios, by NEA code, for application to the TRI emissions data. However, since TRI data are reported by SIC code, we first mapped" NEA codes to the appropriate SIC codes, and used the percent change for each NEA code to represent the percent change for all SIC codes covered by that NEA code.

It should be noted that the Trends data base covers only the most important sources of lead in air, not all sources; as a result, not all SIC codes reporting lead emissions in TRI correspond to an NEA code for which emission differentials have been estimated. However, we assume that the TRI emissions sources that have a match are the most important sources of lead air emissions. In fact, although only 48 out of 519 legitimate SIC codes reporting lead emissions in TRI have matching differentials, these SIC codes account for over 69 percent of the lead emissions reported in TRI. The remaining 31 percent of the emissions are distributed relatively evenly among the remaining 471 SIC codes, each of which contributes a small amount to total emissions.

For the 31 percent of the emissions without differentials, this analysis has no information regarding the change in the lead emissions over time or between the control and no-control scenarios; therefore, we are unable to predict benefits attributable to the CAA for these emission sources. Although excluding these sources may lead us to underestimate total benefits, we believe these sources are unlikely to contribute significantly to the difference between control and no-control scenarios. The Trends data focus on the point sources of lead emissions of greatest concern to the Project Team and of greatest regulatory activity. If a process within an SIC code does not appear in the Trends, it is unlikely to have had specific CAA controls instituted over the past 20 years. A lack of control efficiencies for smaller sources prevents them from being included.

It should also be noted that the total industrial process emissions of lead estimated in the 1990 Trends report actually exceeds the reported lead emissions in

TRI, despite the fact that TRI covers more SIC codes. This is probably attributable in part to the fact that TRI covers only a subset of the facilities contributing to economic output in an SIC code. TRI reporting rules only require facilities with greater than 10 employees and who use certain amounts of lead in their processes to submit information to TRI, while the Trends report attempted to estimate emissions from all sources contributing to the economic output for the industrial sector, regardless of size. However, the components of the Trends data base used in this analysis (i.e., emissions factors, economic output data) represent typical conditions at average facilities; they do not allow for the representation of the distribution of emissions across particular facilities. In contrast, a major strength of the TRI is its match of emissions data with geographical information. Because the distribution of emissions geographically determines the size of exposed populations, this analysis used the TRI data, rather than Trends data, to characterize lead release quantities, and used the Trends figures only to characterize relative emissions and changes over time, rather than to estimate total quantities.

Because the Trends data are intended only as an estimate of emissions using typical conditions at average facilities, and do not capture the differences in facility-level emissions, the data do not provide sufficient information to make specific quantitative adjustments to the TRI-based benefits estimates to account for the overall higher emissions estimates in Trends. However, since Trends does generally suggest that there are many more sources than are accounted for by TRI, it is possible that our benefits calculations may be underestimated.

Some additional assumptions were necessary when matching the TRI lead release data and the differentials from the Trends data. Ideally, we would like to know whether the facilities present at a given location, as reported in the 1990 TRI, were present and operating in earlier years; whether facilities operating in 1970 have ceased to operate; and whether new facilities would have been constructed in the no-control situation. Unfortunately, data do not exist in an accessible form at this level of detail for the years 1970 through 1990. Therefore, for the purposes of this exercise, we have assumed that the locations and numbers of the 1990 sources are the same as they were in 1970.

Methods Used to Determine Changes in Lead Emissions from Industrial Boilers from 1970 to 1990

Several sources were used to determine the change in lead emissions from industrial boilers. TRI locational data, Trends database national fuel consumption levels and emissions factors, and NEA and SIC codes were used to derive the emissions for the control and no-control scenarios.

TRI Data

The TRI does not appear generally to contain combustion emissions data. In general, the emissions data are from process sources. We reached this conclusion based on two pieces of information:

(1) TRI reporting requirements: TRI has three reporting requirements: (a) the facility must fall in SIC codes 20-39; (b) the facility must employ more than 10 persons; and (c) the facility must manufacture or process more than 25,000 pounds of a TRI chemical, or otherwise use more than 10,000 pounds Firms must submit reports only for the chemical that exceeds the thresholds given in item (c), but they must report all releases of that chemical, including releases from uses that would not qualify alone. If the TRI chemical is part of a blended substance and the quantity of the TRI chemical in the blend exceeds the threshold, it must be reported. For industrial boilers, if the amount of lead in the fuel were to exceed the 10,000 pounds threshold, then the firm would be required to report all emissions of lead from combustion of fuel. There is an exemption, however, for ingredients present in small proportions. If the amount of lead in the oil were less than 0.1 percent (1,000 ppm), then the firm would not be required to report the emissions.

The conclusion from the above information is that most firms burning used oil are probably not reporting lead combustion emissions to TRI because these releases fall outside the TRI reporting requirements. The concentration at which lead is typically found is used oil (100 ppm) (NRDC, 1991) is much less than the minimum concentration required for reporting (1,000 ppm).

(2) Use data from the TRI data base: The hypothesis that firms do not report lead combustion was confirmed by an analysis of the data submitted by the

firms reporting lead use to TRI. On the TRI submission forms, firms must indicate how the chemical is used. Our analysis of category codes submitted by firms reporting lead emissions showed the following four use category reports: as a formulation component; as a reactant; as an article component; and repackaging only. None of these category codes suggest that the source of the reported lead release is combustion. Therefore, we may conclude that all of the lead emissions reported in TRI are process emissions.

Based on these analyses, the Project Team could not use the TRI release data to evaluate releases of lead from industrial combustion. However, the TRI geographical information was used to locate industrial facilities by longitude and latitude in order to combine combustion data with population information. For combustion emissions, the calculations included all TRI reporting facilities, not just those who reported lead emissions. The assignment of combustion emissions to these facilities is described below.

Derivation of Industrial Combustion Emissions 1970-1990

As with industrial process emissions, estimates were created for the emissions of lead from industrial combustion under the CAA, and in the absence of the CAA, for the years 1970, 1975, 1980, 1985, and 1990. These emissions estimates were used, in combination with the TRI data base geographic information, to obtain facility-level release estimates for the years of interest for the control and no-control scenarios. The method for deriving these emissions estimates included both the macroeconomic impact of the CAA and the change in emissions per unit of output that resulted from specific pollution control mandates of the CAA. The same data sources were used to derive combustion differentials as were used to derive process differentials. The particular data elements and the methods by which these data were combined to derive lead emissions estimates from industrial combustion are described below.

Estimates of combustion emissions under the control scenario

The Trends database contains a national aggregate industrial fuel consumption estimate, by fuel type (coal, natural gas, oil). For each fuel type, the fuel consumption estimate was disaggregated by the share of that fuel used by each NEA industrial category, using the NEA data base. It should be noted that the NEA includes data only for the years 1970 through 1985. For 1990, the 1985 figures were used to disaggregate the national-level consumption figure into NEA industrial categories.

The Trends database also contains emissions factors for industrial fuel use, by fuel type, as well as control efficiencies. The lead emissions from industrial combustion for each NEA category was derived by multiplying the fuel-specific combustion estimate for each NEA category by the emission factor and control efficiency for that fuel type. The result was emissions of lead by NEA code and by fuel type. Emissions from all fuel types were then summed by NEA code. By using the NEA data to disaggregate the industrial fuel consumption figures, the analysis assumes that the industrial combustion emissions are the same among all industries covered by a given NEA code, an assumption which may bias the analysis.

Estimates of combustion emissions under the no-control scenario

As in the control scenario, the national aggregate industrial fuel consumption estimate, by fuel type (coal, natural gas, oil), was disaggregated by the share of that fuel used by each NEA industrial category. The fuel use was then adjusted by one of two factors: (1) seven of the NEA codes were specifically modeled by the Industrial Combustion Emissions (ICE) model — for these sectors, the ICE modeled percent changes were used instead of J/W percent changes; or (2) the remaining NEA codes were matched to J/W sectors — the J/W percent changes were then applied to those matched NEA codes. These fuel use estimates were then combined with the 1970 emission factors and control efficiencies for industrial combustion by fuel type from the Trends database to obtain combustion-related lead emissions from industrial boilers in the no-control scenario, by NEA code.

The process-specific data in the Trends database, and the energy use data in the NEA, are much more disaggregated than the J/W sectoral projections. For the purpose of the analysis, it was assumed that all of the specific industrial processes in the Trends database and industrial categories in the NEA data set assigned to a given J/W sector changed at the same rate as the entire J/W sector. For example, if the economic activity in the J/W Sector 20, "Primary Metals," changed by one percent between the control and no-control scenarios, then the analysis assumed that economic activity in each industrial process assigned to

the Primary Metals sector also increased by one percent. This approach assumes that the economic activities of specific industries within a sector are equally affected by the imposition of the CAA. This assumption is consistent with the projection of the change in emissions from industrial processes for the other criteria air pollutants, which were calculated using a similar process.

Matching TRI Data to Industrial Combustion Emissions Data

Because of the structure of the TRI reporting requirements, it does not appear that TRI generally contains releases from combustion sources. Although TRI may incidentally contain lead combustion emissions, TRI would contain data on such releases only if the reporting facility also used more than 10,000 pounds of lead per year for manufacturing or processing. As a result, the combustion releases, estimated using the methods described above, do not have corresponding data in the TRI data base. Therefore, we devised a different method for estimating benefits from changes in combustion releases.

The first step in the method was to divide the estimates of total releases of lead from industrial combustion, by NEA code, by an estimate of the number of facilities in each NEA code. The number of facilities in each NEA category was estimated using the 1987 Census of Manufactures. This Census, conducted by the U.S. Department of Commerce, tallies the number of facilities by four-digit SIC code; these SIC codes were matched to the NEA codes.

Dividing total lead emissions emitted by number of facilities yielded the average yearly lead emissions from industrial combustion for each SIC code. We then assigned this average value to *all reporting TRI facilities* in the SIC code. The consequence of this approach is that the modeling of combustion from industrial facilities includes substantially more sources than the modeling of industrial process emissions; combustion emissions are assigned to essentially all facilities reporting to TRI, while the process emissions are only evaluated for facilities actually reporting lead air emissions from processes.

One unavoidable drawback to this approach is that it cannot capture differences in release quantities among facilities within an SIC code. Furthermore, this approach does not capture all combustion emissions because we assign average emissions only to facilities that report to TRI. TRI facilities account for between two percent and 50 percent of all facilities listed in the Census of Manufacturers, depending on the SIC code. Because of the inability to place the remaining facilities geographically, this analysis excludes the consideration of emissions from non-TRI facilities.

Methods Used to Determine Changes in Lead Emissions from Electric Utilities from 1975 to 1990

The estimation of lead emissions from electric utilities required data from three different sources. Energy use data for the control and no-control scenarios were obtained from the national coal use estimates prepared for the section 812 analysis by ICF Incorporated. The OAQPS Trends Database provided emissions factors and control efficiencies. Individual plant latitudes, longitudes, and stack information were collected from the EPA Interim Emissions Inventory. This analysis combines these three sets of data and estimates annual lead emissions at the plant level for coal burning electric utilities in the control and no-control scenarios. This section describes the sources and the methods used to create the final data set.

Coal-Use Data

The energy use data obtained from national coal-use estimates provide plant level energy consumption information for 822 electric utilities. The data set were separated into four distinct sets for the years 1975, 1980, 1985, and 1990. Each set of data provided the state where the plants are located, the plant names, and the amount of coal consumed, for both the control and no-control scenarios. The four data sets were combined into one comprehensive set by matching the plants' names and states.

The EPA Interim Emissions Inventory

The EPA Office of Air Quality Planning and Standards Technical Support Division provided the 1991 EPA Interim Emissions Inventory. The Interim Inventory contains data for all electric utility and industrial plants in the United States including latitude, longitude, stack height, stack diameter, stack velocity, and stack temperature. The additional stack parameter data allowed the use of plant-specific parameters in the air modeling for electric utilities rather than average parameters for all facilities as was done for industrial emissions.

Matching the Coal-Use Data to the Interim Emissions Inventory

The combination of the Interim Emissions Inventory and the coal-use data required two steps. First, the Interim Emissions Inventory had to be pared down to include only electric utility data, and to narrow the information provided for each utility. Second, the two databases had to be combined. One difficulty in combining them was the lack of a common data field that would allow a quick and complete matching process.

Electric utility plants were identified in the Interim Emissions Inventory by SIC code (code 4911). The associated stack information file, which lists the size of every stack on every plant, was reduced to include only the tallest stack for each plant. This provides a reasonable estimate of the stack height at which most emissions occur. The air modeling assumes that each electric utility releases its emissions from the largest stack that exists at that plant.

Next, the procedure matched the abridged Interim Emissions Inventory file with the coal use data. Due to the lack of a common data field between the two sets, this process required several phases. Both data sets had name fields, but these fields utilized different naming conventions for the plants. Therefore the name fields were matched directly, with individual words in the names, and then with abridged words from the names. Abridged word matches were double checked by ensuring that the names were indeed similar and by verifying that the state fields matched. Finally some matches were made by hand.

Only 27 unmatched plants with positive coal use remained. There were 493 matched plants with positive coal usage and these were included in the final data set.[29] To eliminate under-counting of emissions, the emissions from the 27 unmatched plants were allocated to matched plants within the states where the unmatched plants were located. Allocations were weighted according to the emission level for each matched plant within that state in the year in which the allocation was being made.

Emissions Factors and Control Efficiencies

At this stage, the electric utilities data set contained coal consumption by plant by year in the control and no-control cases as well as air modeling parameters. Using emission factors for lead and control efficiencies for electric utilities, estimates of lead emissions per plant per year could now be calculated. As in the industrial source analysis, the emission factors and control efficiencies come from the 1991 OAQPS Trends database.

Control efficiencies are available for coal-fired electric utilities in each year between 1975 and 1990. As in the industrial source analysis, it is assumed that pollution control on coal-burning power plants without the CAA would be the same as the pollution control level in 1970. Therefore, the control efficiency from 1970 is used in the no-control analysis.

The emission factor obtained from the Trends database is expressed in terms of lead emitted per ton of coal burned (6,050 grams per 1,000 tons).[30] The combined data set, though, contains quantity of coal burned per plant per year in energy units (trillions of BTUs). To reconcile this difference, a conversion factor was obtained from a 1992 DOE report titled *Cost and Quality of Fuels for Electric Utility Plants 1991*. The conversion factor used (20.93 million BTUs per ton of coal) is the average BTU per pound of coal burned for all domestic electric utility plants in 1990. Data for a small subset of other years were also provided in the DOE report, but they did not differ significantly from the 1990 number. Therefore, the 1990 conversion factor (637.3 pounds of lead per trillion BTU) is assumed valid over the entire study period. The final equation for lead emissions looks quite similar to the equation used in the industrial source analysis.[31] The only change is that "Economic Activity" has been replaced by "Coal Consumed" for this particular analysis:

$$\textit{Emissions} = \textit{(Coal Consumed)} \times \textit{(Emission Factor)} \times (1 - \textit{Control Efficiency}) \quad (26)$$

This equation produces estimates of the emissions per plant per year in both the control and the no-control scenarios.

[29] Plants with zero coal usage were not immediately excluded from the analysis due to the possibility of analyzing lead emissions from oil combustion at these plants. However, OAQPS has suggested that oil combustion comprises under two percent of the total lead emitted from electric utilities. For this reason, the electric utility analysis focused entirely on coal.

[30] The actual figure cited is 12.1 metric pounds per 1,000 tons. A metric pound is one two-thousandth of a metric ton.

[31] U.S. EPA, 1991a

Use of Air Dispersion Modeling to Estimate Ambient Air Lead Levels

To link estimates of lead emissions to blood lead levels of populations living in the vicinity of a facility, the lead benefits model first uses air dispersion modeling to estimate air lead concentrations surrounding facilities that emit lead into the air. The air concentrations are then linked to blood lead levels.

This analysis uses the Industrial Source Complex Long Term (ISCLT) air dispersion model, a steady-state Gaussian plume model, to estimate long-term lead concentrations downwind of a source. The concentration is modeled as a function of site parameters (stack height, stack velocity).[32] The general form of the concentration equation from a point source at a distance *r* greater than one meter away is as follows:[33]

$$C_{air,r_{ijk}} = \frac{2K}{\sqrt{2\pi}\, r\Theta} \cdot \frac{Q\ f\ S\ V\ D}{u\sigma_z} \qquad (28)$$

where,

- C_{air} = concentration at distance *r* ($\mu g/m^3$),
- Q = pollutant emission rate (g/sec),
- f = frequency of occurrence of wind speed and direction,
- Θ = sector width (radians),
- S = smoothing function used to smooth discontinuities at sector boundaries,
- u = mean wind speed (m/sec),
- σ_z = standard deviation of vertical concentration distribution (m),
- V = vertical term (m),
- K = scaling coefficient for unit agreement.

For each facility modeled in the lead benefits model, a 21 by 21 kilometer grid around the source is specified. The model stores data in 1 km by 1 km cells and calculates the air lead concentrations for each of the 441 cells surrounding a given facility. Fugitive sources are modeled similarly, the only difference being a modified form of Equation 28.

For facility-specific weather data, the model used Stability Array (STAR) data. The STAR data contain information on typical wind speed and direction for thousands of weather stations in the U.S. For each facility, the model accesses the STAR data for the weather station nearest the source. Standard default parameters are used for the other parameters because facility-specific data are not available for them (except for utilities). Table G-3 lists default parameters for the ISCLT, and summarizes sources for other parameters.

Industrial process emissions were modeled as either point or fugitive sources, depending on how they were reported in TRI. All industrial combustion emissions were modeled as "fugitive" emissions. This is a more appropriate model scenario for boiler emissions than a 10 meter stack scenario. All electric utility sources were modeled as point sources.

The model tracks all lead emissions to a given grid cell. That is, if the plumes of two or more sources overlap in a given cell, the air concentration in the given cell is determined from the sum of all of the contributing sources.

Determination of Blood Lead Levels from Air Lead Concentrations

Once the air lead concentrations surrounding a given plant are estimated, the model estimates blood lead levels for children and adults living in those areas. This section describes the methods and data sources used to derive blood lead levels from estimated air lead concentrations.

Relationship Between Air Lead Concentrations and Blood Lead Levels

The rates at which lead is absorbed from air depend on the age of the exposed individual, distance from the facility, the initial concentration of blood lead, and other factors. In addition, rates determined from empirical data may differ depending on whether or not the analyses from which rates are derived have controlled for factors such as lead exposure through deposition on dust and soil (i.e., "indirect exposure"). Especially when children constitute the exposed group, the inclusion of indirect exposure results in higher air lead to blood lead slopes. In both cases, the slope re-

[32] Ideally, reported stack and fugitive air releases would be modeled using site-specific data (such as source area or stack height). However, since TRI does not contain such facility-specific information, default values are used to model TRI facilities.

[33] This equation is from U.S. EPA (1992). The equation is for a specific wind speed, direction, and category (*ijk*). Each facility has several combinations of these that must be added to arrive at a total concentration at that point. The equation for area sources is similar.

Table G-3. Air Modeling Parameters.

Parameter	Industrial Source Value	Electric Utility Value	Source/ Comment
Stack height	10 m	site-specific or 115.0 m*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Exit velocity	0.01 m/s	site-specific or 22.5 m/s*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Stack diameter	1 m	site-specific or 5.15 m*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Exit gas temperature	293° K	site-specific or 427.5*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Area source size	10 m^2	10 m^2	U.S. EPA (1992)
Area source height	3 m	3 m	U.S. EPA (1992)
Lead emission rate	site-specific	site-specific	Industrial – TRIS (lbs/yr) Utilities -- SAI & OAQPS (lbs/yr)
Frequency of wind speed and direction	site-specific	site-specific	STAR data
Sector width	22.5°	22.5°	360° divided by 16 wind directions
Wind speed	site-specific	site-specific	STAR data (m/sec)
Smoothing function	calculated	calculated	
Vertical term	calculated	calculated	

* average value for electric utilities, utilized for utilities without this information

lationship is expressed as the change in blood lead (µg/dL) per change in air concentration (µg/m^3).

In performing this analysis, a choice had to be made between the use of air lead:blood lead relationships that account for inhalation exposure ("direct" slopes) and those that account for exposure to lead deposited from air onto soil and dust ("indirect" slopes). The choice of which slopes to use considered both the effects on the estimate of benefits over time (from 1970 to 1990) and the estimate of the difference in benefits between the control and no-control scenarios. The indirect slope is more comprehensive in its coverage of the types of exposures that will result from air releases, and thus captures more of the health effects predicted to occur from lead exposures, especially to children. For this reason, indirect slopes are preferred to direct slopes, especially when comparing the control and no-control scenarios: using only the direct slope would underestimate the benefits of avoiding deposition that controls confer. However, indirect slopes may capture effects from exposure to soil and dust lead deposited from both current air releases and historic air releases. Since lead's dissipation from soil is slow relative to its removal from air, the reservoir of lead in soil and dust is unlikely to change at the same rate as the reductions in air lead concentrations. Therefore, using indirect slopes to represent a change in blood lead over time due to reduced air emissions may overestimate the change in blood lead, and thus overestimate the benefits of reductions over time, to the extent that the indirect slope captures exposure to the total reservoir of soil and dust lead, rather than only recently deposited lead.

Given that the focus of this analysis is the *difference* between the control and no-control scenarios, it is important to capture both the benefits from reduced lead deposition that result from the CAA, and the direct benefits from reduced air concentrations. Therefore, this analysis modeled changes in blood lead levels using indirect slopes. It should be kept in mind

that this choice may overestimate blood lead changes over time for both the control and no-control scenarios.

The relationship between concentrations of lead in ambient air and blood lead concentrations has been evaluated by a variety of methods. These include experimental studies of adult volunteers, as well as epidemiological studies of different populations of children and adults. The discussion below describes the slopes used in this analysis for children and adults, and for individuals with blood lead values greater than 30 µg/dL.

Children

U.S. EPA (1986b) reports that slopes which include both direct (inhalation) and indirect (via soil, dust, etc.) air lead contributions vary widely, but typically range from three to five µg/dL increment in children's blood lead per µg/m^3 increment in air lead concentration (roughly double the slope due to inhaled air lead alone). Since hand dust levels can play a significant role in blood lead levels (U.S. EPA, 1986b), this higher slope may be due to mouthing behavior of children that brings them into contact with dust and soil.

Specific values for estimating contribution of air lead to blood lead, including indirect pathways, are cited in U.S. EPA (1986b); slope values (ranging from -2.63 to 31.2) and data sources for these values are presented in Table 11-36 of U.S. EPA (1986b). The median of these values is 4.0 µg/dL per µg/m^3, which matches the midpoint of the range of typical slope values. This analysis used this value to represent the relationship between air lead concentrations and blood lead concentrations for children living in the vicinity of point sources of lead emissions.

The use of this slope assumes that indirect exposure" principally measures indirect effects of lead emissions to air (through deposition to dust and soil). However, it is possible that these slopes include other exposures not related to air lead. In many cases researchers have measured other possible exposures, such as water and food, and have confirmed that the most significant contribution comes from soil and dust lead, which is assumed to result from air deposition of lead. Those studies that measured lead in tap water showed that mean levels were generally low or not significantly related to blood lead. Landrigan et al. (1975) measured lead in pottery and food; lead in pottery was found in only 2.8 percent of homes, and food and water made no more than a negligible contribution to lead uptake. Lead in paint was measured in some studies.[34] Landrigan and Baker (1981) measured lead in paint at levels greater than one percent in about one fourth to one third of the houses in each area studied. Brunekreef et al. (1981) measured high levels of paint in some houses, but excluded these data points from the analysis.

Despite the possibility of confounding factors, this analysis uses the median value determined above (4.0 µg/dL per µg/m^3) as the appropriate slope for children living within five kilometers of the point source. Five kilometers is chosen as the cut off point because the data from most of the studies cited collected the majority of their data points near lead smelters.[35] Furthermore, these slopes, although measured primarily in the vicinity of smelters, are assumed applicable to all point sources that emit lead into the air.

Adults

For adult males and females, the air lead/ blood lead slopes that include indirect effects due to soil and dust differ very little from slopes that include only direct effects. This result is expected since the higher indirect slope values estimated for children are assumed to be as a result of mouthing behavior typical of young children.

U.S. EPA (1986b) describes several population studies that estimate indirect slopes for men; these slopes range from -0.1 to 3.1 µg/dL per µg/m^3.[36] Snee (1981) determined a weighted average of these studies and one other study.[37] The average slope, weighted by the inverse of each study's variance, is 1.0 µg/dL per µg/m^3. However, the Azar study measured the direct relationship between air lead and blood lead. Excluding the Azar study from the weighted average, the average slope is 1.1 µg/m^3. Excluding the highest and lowest slopes from this group (from Goldsmith,

[34] Landrigan and Baker, 1981; Brunekreef et al., 1981.

[35] U.S. EPA, 1986b, Table 11-36.

[36] Johnson et al., 1976; Nordman, 1975; Goldsmith, 1974; Tsuchiya et al., 1975; Fugas et al., 1973.

[37] Azar et al., 1975.

1974 and Tsuchiya et al., 1975), both of which had difficulties,[38] the resulting slope is 1.4 µg/dL per µg/m³.

Slopes for females range from 0.6 to 2.4 for general atmospheric conditions.[39] Snee determined an average slope for women of 0.9 µg/dL per µg/m³, weighted by the inverse of the variances of the studies. Excluding the slope for women from Goldsmith (1974), the resulting slope for women is 1.0 µg/dL per µg/m³.

These values are adjusted by a factor of 1.3 to account for the resorption of lead from bone tissue (according to Chamberlain, 1983), thus deriving an adjusted slope estimate of 1.8 µg/dL blood lead per µg/m³ increment in air lead concentration for men and 1.3 for women. These are the slope estimates used in this analysis.

Individuals with initial blood lead levels of 30 µg/dL and greater

For individuals with high blood lead levels, the air lead to blood lead uptake slopes have been shown to be much shallower, as described by U.S. EPA (1986b). An appropriate change in blood lead per change in air lead is 0.5 µg/dL per µg/m³ for individuals that have initial blood lead levels in the range of 30 to 40 µg/dL. This value is based on cross-sectional and experimental studies.[40] For individuals with initial blood lead levels greater than 40 µg/dL, an appropriate range of slopes is 0.03 to 0.2, as determined by occupational studies listed in Table 11-37 of U.S. EPA (1986b). The median value of these studies is 0.07. These two slopes (0.5 for the population with blood lead levels between 30 and 40 µg/dL and 0.07 for blood lead levels greater than 40 µg/dL) are used for both children and adults in this analysis. These relationships are summarized in Table G-4.

Estimates of Initial Blood Lead Concentrations

The benefits model requires an initial distribution of blood lead levels in the exposed populations to model health benefits of reducing lead air emissions. The model estimates the new distribution of blood lead levels that would exist after a given change in air concentrations using the slopes described above. Finally, the model estimates the difference between the two distributions. This analysis begins with an initial 1970, no-control scenario blood lead distribution from which all subsequent changes are modeled. This approach requires an estimate of the blood lead distributions in the U.S. population in 1970. Unfortunately, there are no actual national blood lead distribution estimates for 1970. Although the first NHANES study covered 1970, blood lead data were not collected in this study.[41] Nonetheless, a 1970 distribution of blood lead was estimated using NHANES II data (from 1976-1980), combined with estimates of typical changes in blood lead levels from 1970-1976 observed in localized screening studies.

Table G-4. Estimated Indirect Intake Slopes: Increment of Blood Lead Concentration (in µg/dL) per Unit of Air Lead Concentration (µg/m³).

	Individuals with blood lead levels < 30 µg/dL	Individuals with blood lead levels 30-40 µg/dL	Individuals with blood lead levels > 40 µg/dL
Adult Males	1.8	0.5	0.07
Adult Females	1.3	0.5	0.07
Children	4.0	0.5	0.07

[38] Goldsmith (1974) refrigerated (rather than froze) the blood samples, and did not analyze the samples until 8 or 9 months after they were taken, and restricted the analysis to one determination for each blood sample. Tsuchiya et al. (1975) measured air lead concentrations after blood samples were taken; blood was drawn in August and September of 1971, whereas air samples were taken during the 13 month period from September 1971 to September 1972.

[39] Tepper and Levin, 1975; Johnson et al., 1976; Nordman, 1975; Goldsmith, 1974; Daines et al., 1972.

[40] U.S. EPA, 1986b.

[41] NCHS, 1993a.

A major drawback to this approach is the uncertainty in deriving the 1970 estimates. Another drawback to beginning with the 1970 level and modeling changes from that point is the analysis only represents changes in lead exposure from air; reductions from other sources of lead exposure are not accounted for. The purpose of this analysis is to identify changes attributable to the CAA mandates; changes from other sources of lead exposure should not be considered. However, due to nonlinear nature of the lead concentration-response functions (see above), the overall exposure context in which the air lead exposure reductions take place will influence the estimate of benefits from those reductions. Specifically, at higher blood lead levels, the slope of the concentration-response curve is shallower than at lower levels. As a result, a given change in the mean blood lead level may result in a smaller change in the health effect if the change occurs from a relatively high starting level. On the other hand, if one accounts for the fact that other sources of lead exposure are reduced at the same time that the given air reductions occur, then those air emissions reductions may result in greater changes in health risk.

This issue is of concern even though the analysis focuses on the difference between the control and no-control scenarios, since the health benefit implications of the emissions differentials between the scenarios will depend on the point on the blood lead distribution curve at which the differences are considered. That is, a difference between a mean blood lead of 25 µg/dL and one of 20 µg/dL may have different health implications than a difference between 15 µg/dL and 10 µg/dL, even though the absolute value of the difference is the same (5 µg/dL).

An alternative method is to "start" with a 1990 blood lead level and to "back-calculate" benefits by representing the differentials as increases over the 1990 levels, rather than decreases from 1970 levels. The advantage of this approach is that it accounts for reductions in lead exposure from other sources, as represented by current blood lead levels. Its disadvantage is that it holds other sources constant to (lower) 1990 levels, and thus the modeling may underestimate actual blood lead distributions in earlier years, and thereby overestimate benefits from controls during those years. This analysis presents the results of both approaches, indicated as "forward-looking" and "backward-looking".

Combination of Air Concentration Estimates with Population Data

The modeled air lead concentrations at various distances from the sources were combined with population data from the Census Bureau to arrive at an estimate of the number of cases of health effects for each of the years from 1970 to 1990 in both the control and no-control scenarios. The primary census information was accessed from the Graphical Exposure Modeling System Database (GEMS), an EPA mainframe database system. The following data were obtained from GEMS for the years 1970, 1980, and 1990: total population for each Block Group/Enumeration District (BG/ED); state and county FIPS codes associated with each BG/ED; latitude and longitude of each BG/ED; and population of males under 5 and females under 5 for each BG/ED. The intervening five year intervals (1975 and 1985) were estimated using the Intercensal County Estimates from the Census, which estimate annual populations on a county by county basis. The decennial Census data and the Intercensal County Estimates data sets were related by county FIPS codes; the population in each BG/ED was assumed to grow or shrink at the same rate as the county population as a whole.

Since the concentration-response data are particular to specific sex and adult age groups, additional population data were also required to determine the sizes of affected subpopulations. For 1990 age and sex, the U.S. Census, 1992 was used, with age groups tallied as necessary. For 1980 age and sex, the U.S. Census, 1982 was used, with age groups also tallied as necessary. The 1970 age and sex breakdowns were obtained through personal communication with the Census Bureau.[42] The age and sex percentages were interpolated for intervening years.

Pregnant women are often a subpopulation of interest for lead effects. Although pregnant women themselves may be harmed by exposure to lead, this analysis was concerned with pregnant women because of possible effects on their fetuses who will be born

[42] Personal communication, Karl Kuellmer, Abt Associates and the Bureau of Census, Population, Age and Sex telephone staff, March, 1994.

and evince effects as young children. To estimate the number of exposed fetuses who were born during the years of interest,[43] birth rates for 1970, 1980 and 1990 were obtained from the Census Bureau.[44] These birth rates were used to interpolate for years between 1970 and 1980, and for the years between 1980 and 1990.

Results

For both the control and no-control scenarios, Table G-5 shows estimated lead emissions from electric utilities, industrial processes, and industrial combustion. Tables G-6 and G-7 show the differences in health impacts between the two scenarios (for industrial processes, industrial combustion and electric utilities only) for the "forward-looking" and "backward-looking" analyses. The modeled population for each year is also presented.

Table G-5. Estimated Lead Emissions from Electric Utilities.Industrial Processes.and Industrial Combustion (in Tons).

	1970	1975	1980	1985	1990
Electric Utilities[a] Control Scenario		1,351	636	175	190
Electric Utilities[a] No-control Scenario		2,309	3,143	3,670	3,864
Industrial Processes Control Scenario	7,789	3,317	1,032	670	658
Industrial Processes No-control Scenario	7,789	7,124	6,550	5,696	5,305
Industrial Combustion Control Scenario	4,329	4,354	1,880	190	187
Industrial Combustion No-control Scenario	4,329	4,457	4,653	4,584	4,596

[a] Appropriate data on electric utilities do not exist for years prior to 1975.

[43] Note that we do not record the number of pregnancies, since the valuation only applies if the child is born and lives to exhibit the effect. Neither are we concerned with whether the births are single or multiple births, since each fetus is at risk, whether a pregnant women carries one or more fetuses.

[44] Personal communication, Karl Kuellmer, Abt Associates and the Bureau of Census, Population, Fertility/Births telephone staff.

Table G-6. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1970 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	0.1	1.5	2.5	2.7
Men (55-64)	0.0	1.1	1.8	1.8
Men (65-74)	0.0	0.4	0.7	0.8
Women (45-74)	0	0.8	1.3	1.4
Infants	0	0.001	0.002	0.002
Total	0.1	3.9	6.3	6.7
Coronary Heart Disease				
Men (40-54)	0.1	1.8	3.0	3.3
Men (55-64)	0.0	0.7	1.2	1.2
Men (65-74)	0.0	1.0	1.6	1.7
Women (45-74)	0.1	1.3	2.1	2.1
Total	0.2	4.8	8.0	8.3
Strokes				
Cerebrovascular Accident (men 45-74)	0.1	1.1	1.8	1.8
Cerebrovascular Accident (women 45-74)	0	0.5	0.9	0.9
Brain Infarction (men 45-74)	0	0.7	1.1	1.1
Brain Infarction (women 45-74)	0	0.4	0.6	0.6
Total	0.1	2.7	4.4	4.4
Hypertension (men 20-74)	149	3,790	6,350	6,670
IQ Decrement				
Lost IQ Points	630	14,300	22,700	23,900
IQ<70 (cases)	3	60	120	125
Population Exposed (millions)	188	197	207	217

Table G-7. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1990 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	0.3	6.9	11.5	12.5
Men (55-64)	0.2	5.1	8.3	8.2
Men (65-74)	0.1	2.0	3.5	3.9
Women (45-74)	0.2	3.9	6.4	6.4
Infants	0	0.001	0.002	0.002
Total	0.8	17.9	29.7	31.0
Coronary Heart Disease				
Men (40-54)	0.4	8.3	13.8	15.0
Men (55-64)	0.1	3.4	5.6	5.6
Men (65-74)	0.2	4.4	7.6	8.0
Women (45-74)	0.2	5.9	9.6	9.7
Total	0.9	22.1	36.6	38.3
Strokes				
Cerebrovascular Accident (men 45-74)	0.2	5.0	8.1	8.2
Cerebrovascular Accident (women 45-74)	0.1	2.6	4.1	4.2
Brain Infarction (men 45-74)	0.1	2.8	4.6	4.7
Brain Infarction (women 45-74)	0.1	1.6	2.7	2.7
Total	0.5	12.0	19.5	19.8
Hypertension (men 20-74)	422	10,800	18,100	19,000
IQ Decrement				
Lost IQ Points	630	14,300	22,700	23,900
IQ<70 (cases)	0	31	50	61
Population Exposed (millions)	188	197	207	217

Reduction in Health Effects Attributable to Gasoline Lead Reductions

Estimating Changes in Amount of Lead in Gasoline from 1970 to 1990

The relationship between the national mean blood lead level and lead in gasoline is calculated as a function of the amount of lead in gasoline consumed. Thus, to calculate the health benefits from gasoline lead reductions, necessary inputs are estimates of lead in gasoline consumed over the period 1970 to 1990 and the amount of lead in gasoline that would have been consumed in the absence of the Clean Air Act. These values are calculated using the quantity of both leaded and unleaded gasoline sold each year and the concentration of lead in leaded and unleaded gasoline for each year in the period of interest. For each year, the relationship is expressed as:

$$LEAD = \left(\frac{SOLD}{365\ days}\right) \times [FRAC_{Pb} \times PB_{leaded} + (1 - FRAC_{Pb}) \times PB_{unleaded}] \quad (29)$$

where:

- $LEAD$ = average lead per day in gasoline sold in a given year (metric tons/day),
- $SOLD$ = total quantity of gasoline sold (million gal/yr),
- $FRAC_{Pb}$ = fraction of total gasoline sales represented by leaded gasoline (dimensionless),
- Pb_{leaded} = lead content of leaded gasoline (g/gal), and
- $Pb_{unleaded}$ = lead content of unleaded gasoline (g/gal).

Gasoline Sales (SOLD): Data on annual gasoline sales were taken from a report by Argonne National Laboratories (1993) which presented gasoline sales for each state in five year intervals over the period 1970-1990. This analysis used linear interpolation to estimate the gasoline sales for years between the reported years. These data were summed to obtain national sales figures.

Fraction of Total Sales Comprised of Leaded Gasoline ($FRAC_{Pb}$): For the control scenario, this analysis used information reported by Kolb and Longo (1991) for the fraction of the gasoline sales represented by leaded gasoline for the years 1970 through 1988. For 1989 and 1990, data were taken from DOE (1990 and 1991, respectively). For the no-control scenario, all of the gasoline sold was assumed to be leaded for all years.

Lead Content of Gasoline (Pb_{leaded} and $Pb_{unleaded}$): Argonne National Laboratory in Argonne, Illinois was the source for the data on the lead content of leaded and unleaded gasoline for the period 1974-1990. Argonne compiled these data from historical sales data submitted to EPA, from Clean Air Act regulations on lead content, and from recent Motor Vehicle Manufacturers Association (MVMA) surveys. For 1970 through 1973, this analysis assumed the lead content of gasoline to be at the 1974 level. For the no-control scenario, this analysis used the 1974 lead content in leaded gasoline as the lead content in all gasoline for each year.

Estimating the Change in Blood Lead Levels from the Change in the Amount of Lead in Gasoline

Several studies have found positive correlations between gasoline lead content and blood lead levels.[45] Data from the National Health and Nutrition Examination Survey (NHANES II) have been used by other researchers who determined similar positive correlations between gasoline lead and blood lead levels.[46]

The current analysis used a direct relationship between consumption of lead in gasoline and blood lead levels to estimate changes in blood lead levels resulting from Clean Air Act regulation of the lead content of gasoline. This relationship was based on regression analyses of the reduction of leaded gasoline presented in the 1985 Regulatory Impact Analysis (RIA).[47] Several multiple regressions were performed in the RIA to relate gasoline usage with individuals' blood lead lev-

[45] U.S. EPA, 1985; Billick et al., 1979; Billick et al., 1982.

[46] Janney, 1982; Annest et al., 1983; Centers for Disease Control, 1993; National Center for Health Statistics, 1993b.

[47] U.S. EPA, 1985.

els, which were taken from NHANES II. These regressions of blood lead on gasoline usage controlled for such variables as age, sex, degree of urbanization, alcohol consumption, smoking, occupational exposure, dietary factors, region of the country, educational attainment, and income. The regressions suggested that a decrease of 100 metric tons per day (MTD) of lead used in gasoline is associated with a decrease in mean blood lead concentration of 2.14 µg/dL for whites and 2.04 µg/dL for blacks. In both of these regressions, gasoline use was found to be a highly significant predictor of blood lead ($p < 0.0001$).[48]

To determine a single gasoline usage-blood lead slope for the entire population of the U.S., this analysis used the average of the slopes for blacks and for whites, weighted by the percentage of blacks and whites in the U.S. during the time period of the analysis.[49] The resulting relationship is 2.13 µg/dL blood lead per 100 metric tons of lead in gasoline consumed per day. The same relationship was used to model changes in both children's and adults' blood lead levels. The U.S. EPA (1985) analyzed data from a study of black children in Chicago during the time period 1976 to 1980 and determined a slope of 2.08 µg/dL per 100 MTD. This slope for children is very similar to the one used in this analysis.

1970-Forward and 1990-Backward Approaches

As with the industrial processes and boilers analysis, this analysis used two different approaches to determine mean blood lead levels based on changes in lead concentrations in gasoline. In the 1970-forward approach, the calculations began with the estimated blood lead level for 1970. The change in blood lead level from one year to the next was based upon the change in the amount of lead in gasoline sold, as discussed above, for both the control and no-control scenarios. For example, to calculate the blood lead level for 1971, the calculated change in blood lead from 1970 to 1971 was added to the 1970 value. This process was repeated for each succeeding year up to 1990.

The 1990-backward approach began with a mean blood lead level in 1990 for the control scenario. For the no-control scenario, the starting blood lead was estimated from the 1990 level used in the control scenario, plus an additional blood lead increment resulting from the difference between the 1990 consumption of lead in gasoline under the two scenarios. Again, the difference in mean blood lead levels from one year to the next was based on the change in gasoline lead for the corresponding years. For example, the difference in blood lead levels between 1990 and 1989 was subtracted from the 1990 level to determine the 1989 level. The process was continued for each year back to 1970.

Relating Blood Lead Levels to Population Health Effects

The mean blood lead levels calculated using the methods described above were used in the dose-response functions for various health effects (e.g., hypertension, chronic heart disease, mortality). This information was then combined with data on the resident population of the 48 conterminous states in each year to determine the total incidence of these health effects attributable to lead in gasoline. A Department of Commerce Publication (1991) was used to obtain the total population in 1970, 1980, and 1983-1990, while a different publication was the source of the 1975 population values.[50] Linear interpolation was used to estimate the populations in years for which specific data were not available.

For certain health effects, it was necessary to know the size of various age groups within the population. Two different sources were used to estimate the proportions of the population in the age groups of interest. A U.S. Census summary (U.S. Dept. of Commerce, 1990) was used for information for 1990 for children and adults and for 1980 for adults, and Census Telephone Staff (U.S. Dept. of Commerce, 1994) provided information for 1980 for children and 1970 for children and adults. The populations for the intervening years were estimated by linear interpolation.

Changes in Leaded Gasoline Emissions and Resulting Decreased Blood Lead Levels and Health Effects

Table G-8 shows the estimated quantity of lead burned in gasoline in the five year intervals from 1970 to 1990. Tables G-9 and G-10 show the difference in

[48] U.S. EPA, 1985.

[49] U.S. Department of Commerce, 1992. Although the percentages of blacks and whites changed slightly over this time period (1970-1990), the change did not affect the value of the weighted slope.

[50] U.S. Dept. of Commerce, 1976.

health impacts between the two scenarios (for lead in gasoline only) for the "forward-looking" and "backward-looking" analyses. In general, health effect benefits resulting from gasoline lead reductions exceed those predicted from lead reductions at the point sources examined (i.e., industrial processes and boilers and electric utilities) by three orders of magnitude.

Table G-8. Lead Burned in Gasoline (in tons).

	1970	1975	1980	1985	1990
Control Scenario	176,100	179,200	86,400	22,000	2,300
No-control Scenario	176,100	202,600	206,900	214,400	222,900

Table G-9. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1970 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	309	1,820	3,340	4,150
Men (55-64)	220	1,340	2,380	2,700
Men (65-74)	81	520	999	1,260
Women (45-74)	155	939	1,710	2,060
Infants	456	2,340	3,930	4,940
Total	1,220	6,960	12,400	15,100
Coronary Heart Disease				
Men (40-54)	230	1,360	2,540	3,280
Men (55-64)	92	563	1,030	1,220
Men (65-74)	113	723	1,380	1,750
Women (45-74)	73	442	805	965
Total	508	3,090	5,760	7,210
Strokes				
Cerebrovascular Accident (men 45-74)	147	884	1,610	1,960
Cerebrovascular Accident (women 45-74)	73	442	805	965
Brain Infarction (men 45-74)	85	508	927	1,130
Brain Infarction (women 45-74)	47	286	521	624
Total	352	2,120	3,862	4,679
Hypertension (men 20-74)	677,000	4,200,000	7,840,000	9,740,000
IQ Decrement				
Lost IQ Points	1,030,000	5,020,000	8,580,000	10,400,000
IQ<70 (cases)	3,780	20,100	36,500	45,300
Population Exposed (millions)	214	225	237	247

Table G-10. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1990 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	476	3,040	6,140	7,950
Men (55-64)	342	2,250	4,430	5,240
Men (65-74)	128	886	1,880	2,480
Women (45-74)	242	1,590	3,210	4,030
Infants	456	2,340	3,930	4,940
Total	1,640	10,100	19,600	24,600
Coronary Heart Disease				
Men (40-54)	356	2,280	4,690	6,310
Men (55-64)	142	945	1,910	2,370
Men (65-74)	176	1,220	2,570	3,380
Women (45-74)	113	740	1,490	1,860
Total	787	5,180	10,700	13,900
Strokes				
Cerebrovascular Accident (men 45-74)	225	1,460	2,940	3,720
Cerebrovascular Accident (women 45-74)	113	740	1,490	1,860
Brain Infarction (men 45-74)	129	837	1,680	2,120
Brain Infarction (women 45-74)	73	477	955	1,190
Total	540	3,514	7,065	8,890
Hypertension (men 20-74)	984,000	6,350,000	12,300,000	15,600,000
IQ Decrement				
Lost IQ Points	1,030,000	5,030,000	8,580,000	10,400,000
IQ<70 (cases)	3,790	20,100	36,500	45,300
Population Exposed (millions)	214	225	237	247

Lead Benefits Analysis References

Abt Associates, Inc. 1992. *The Medical Costs of Five Illnesses Related to Exposure to Pollutants*. Prepared for: Nicholas Bouwes, Regulatory Impacts Branch, Economics and Technology Division, Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency, Washington, D.C.

Abt Associates, Inc. 1995. *The Impact of the Clean Air Act on Lead Pollution: Emissions Reductions, Health Effects, and Economic Benefits From 1970 to 1990, Draft.* Prepared for Economic Analysis and Innovations Division, Office of Policy Planning and Evaluation, U.S. EPA. January 19.

Annest, J.L., J.L. Pirkle, D. Makuc, J.W. Neese, D.D. Bayse, and M.G. Kovar. 1983. "Chronological Trend in Blood Lead Levels Between 1976 and 1980." *New England Journal of Medicine* 308: 1373-1377.

Argonne National Laboratories (Argonne). 1993. National Gasoline Sales Data, 1970-1990.

Ashenfelter, O. and J. Ham. 1979. "Education, Unemployment and Earnings." *J. Political Economy* 87(5): S99-S131.

Azar, R.D., et al. 1975. An Epidemiologic Approach to Community Air Lead Exposure Using Personal Air Samplers. In: Griffin, T.B. and Knelson, J.H., eds. Lead. Stuttgart, West Germany: Georg Thieme Publishers; pp.254-290. (Coulston, F. and Korte, F., eds. Environmental Quality and Safety: Supplement V. 2).

Bellinger, D., J. Sloman, A. Leviton, M. Rabinowitz, H.L. Needleman, and C. Waternaux. 1991. "Low-level Lead Exposure and Children's Cognitive Function in the Preschool Years." *Pediatrics* 87(2): 219-227.

Bellinger, D.C. 1992. "Lead Exposure, Intelligence and Academic Achievement. *Pediatrics* 90(6): 855.

Billick, I.H., A.S. Curran, and D.R. Shier. 1979. "Analysis of Pediatric Blood Lead Levels in New York City for 1970-1976." *Environmental Health Perspectives* 31: 183-190.

Billick, I.H., et al. 1982. *Predictions of Pediatric Blood Lead Levels from Gasoline Consumption*. U.S. Department of Housing and Urban Development. [Cited in U.S. EPA, 1985.]

Brunekreef, B.D., et al. 1981. "The Arnhem Lead Study: 1. Lead Uptake by 1- to 3-year-old Children Living in the Vicinity of a Secondary Lead Smelter in Arnhem, the Netherlands." *Environ. Res.* 25: 441-448.

Centers for Disease Control (CDC). 1985. *Preventing Lead Poisoning in Young Children.* U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Atlanta, GA.

Centers for Disease Control (CDC). 1991a. *Strategic Plan for Elimination of Childhood Lead Poisoning.* U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Atlanta, GA. February.

Centers for Disease Control (CDC). 1991b. *Preventing Lead Poisoning in Young Children.* U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Atlanta, GA. October.

Centers for Disease Control (CDC). 1993. Personal communication between Abt Associates and Jim Pirkle. November 16.

Chamberlain, A.C. 1983. "Effect of Airborne Lead on Blood Lead." *Atmos. Environ.* 17: 677-692.

Daines, R.H., et al. 1972. "Air Levels of Lead Inside and Outside of Homes." *Ind. Med. Surg.* 41: 26-28.

Dietrich, K.N., K.M. Krafft, R. Shukla, R.L. Bornschein, and P.A. Succop. 1987. The Neurobehavioral Effects of Prenatal and Early Postnatal Lead Exposure. In: *Toxic Substances and Mental Retardation: Neurobehavioral Toxicology and Teratology,*

S.R. Schroeder, Ed. American Association of Mental Deficiency, Washington, DC, pp. 71-95 (Monograph No. 8).

Elixhauser, A., R. M. Andrews, and S. Fox. 1993. *Clinical Classifications for Health Policy Research: Discharge Statistics by Principal Diagnosis and Procedure.* Agency for Health Care Policy and Research, Center for General Health Services Intramural Research, U.S. Department of Health and Human Services.

Environmental Law Institute (ELI). 1992. Projecting With and Without Clean Air Act Emissions for the Section 812 Retrospective Analysis: A Methodology Based upon the Projection System Used in the OAQPS "National Air Pollutant Emission Estimates: Reports." [Jorgenson/Wilcoxen Model Projections], Jim Lockhart.

Fugas, M., et al. 1973. "Concentration Levels and Particle Size Distribution of Lead in the Air of an Urban and an Industrial Area as a Basis for the Calculation of Population Exposure." In: Barth, D., et al. eds. Environmental Health Aspects of Lead: Proceedings, International Symposium; October 1972; Amsterdam, The Netherlands. Luxembourg: Commission of the European Communities, pp. 961-968.

Goldsmith, J.R. 1974. Food Chain and Health Implications of Airborne Lead. Sacramento, CA: State of California, Air Resources Board; Report No. ARB-R-102-74-36.

Griliches, Zvi. 1977. "Estimating the Returns to Schooling: Some Econometric Problems." *Econometrica* 45:1-22.

Hasselblad, V. 1995. Personal Communication between V. Hasselblad and Abt Associates, February 28, 1995.

Janney, A. 1982. *The Relationship Between Gasoline Lead Emissions and Blood Poisoning in Americans.* Prepared for U.S. EPA, Office of Policy Analysis. [Cited in U.S. EPA, 1985.]

Johnson, D.E., et al. 1976. *Base Line Levels of Platinum and Palladium in Human Tissue.* U.S. EPA, Health Effects Research Laboratory, Research Triangle Park, N.C. EPA-600/1-76-019.

Kakalik, J., et al. 1981. *The Cost of Special Education.* Rand Corporation Report N-1791-ED.

Kolb, J. and K. Longo. 1991. Memorandum to Joel Schwartz, U.S. EPA, Washington, DC, November 5.

Krupnick, A.J. and M.L. Cropper. 1989. *Valuing Chronic Morbidity Damages: Medical Costs and Labor Market Effects.* Draft Final Report to U.S. Environmental Protection Agency, Office of Policy Planning and Evaluation. June 26.

Landrigan, P.J., et al. 1975. "Epidemic Lead Absorption near an Ore Smelter: the Role of Particulate Lead." *N. Engl. J. Med.* 292: 123-129.

Landrigan, P.J. and E.L. Baker. 1981. "Exposure of Children to Heavy Metals from Smelter: Epidemiology and Toxic Consequences." *Environ. Res.* 25: 204-224.

McGee and Gordon. 1976. The Results of the Framingham Study Applied to Four Other U.S.-based Epidemiologic Studies of Coronary Heart Disease. *The Framingham Study: An Epidemiological Investigation of Cardiovascular Disease*. Section 31, April.

National Center for Health Statistics (NCHS). 1993a. Facsimile received by Abt Associates from Margaret McDowell regarding the types of laboratory tests conducted during NHANES I. December 14.

National Center for Health Statistics (NCHS). 1993b. Personal communication between Abt Associates and NCHS Public Information Specialist. November 3.

National Energy Accounts, Bureau of Economic Analysis.

Needleman, H.L., et al. 1990. "The Long Term Effects of Exposure to Low Doses of Lead in Children." *New England Journal of Medicine* 322(2): 83-88.

NHANES, National Health and Nutrition Examination Survey.

NHANES II, National Health and Nutrition Examination Survey, 1976-1980.

Nordman, C.H. 1975. *Environmental Lead Exposure in Finland: a Study on Selected Population Groups* [dissertation]. Helsinki, Finland: University of Helsinki.

Oliver, T. 1911. "Lead Poisoning and the Race." *British Medical Journal* 1(2628): 1096-1098. [Cited in USEPA (1990).]

Piomelli et al. 1984. "Management of Childhood Lead Poisoning." *Pediatrics* 4: 105.

Pirkle, J.L., J. Schwartz, J.R. Landis, and W.R. Harlan. 1985. "The Relationship Between Blood Lead Levels and Blood Pressure and its Cardiovascular Risk Implications." *American Journal of Epidemiology* 121: 246-258.

Pirkle, J. L., et al. 1994. "Decline in Blood Lead Levels in the United States, the National Health and Nutrition Examination Survey (NHANES)." *JAMA* 272(4): 284.

Pooling Project Research Group. 1978. "Relationship of Blood Pressure, Serum Cholesterol, Smoking Habit, Relative Weight and ECG Abnormalities to Incidence of Major Coronary Events: Final Report of the Pooling Project." *Journal of Chronic Disease*. Vol. 31.

Rabinowitz, M., D. Bellinger, A. Leviton, H. Needleman, and S. Schoenbaum. 1987. "Pregnancy Hypertension, Blood Pressure During Labor, and Blood Lead Levels." *Hypertension* 10(4): October.

Salkever, D.S. 1995. "Updated Estimates of Earnings Benefits from Reduced Exposure of Children to Environmental Lead." *Environmental Research* 70: 1-6.

Schwartz, J. 1988. "The Relationship Between Blood Lead and Blood Pressure in the Nhanes II Survey." *Environmental Health Perspectives*. 78: 15-22.

Schwartz, J. 1990. "Lead, Blood Pressure, and Cardiovascular Disease in Men and Women." *Environmental Health Perspectives*, in press.

Schwartz, J. 1992a. "Blood Lead and Blood Pressure: a Meta-analysis." Presented at the *Annual Meeting of Collegium Ramazzini.* November.

Schwartz, J. 1992b. "Chapter 13: Lead, Blood Pressure and Cardiovascular Disease." In: *Human Lead Exposure*, H. L. Needleman, Ed. CRC Press.

Schwartz, J. 1993. "Beyond LOEL's, p Values, and Vote Counting: Methods for Looking at the Shapes and Strengths of Associations." *Neurotoxicology* 14(2/3): October.

Shurtleff, D. 1974. Some Characteristics Related to the Incidence of Cardiovascular Disease and Death. *The Framingham Study: An Epidemiological Investigation of Cardiovascular Disease*. Section 30, February.

Silbergeld, E.K., J. Schwartz, and K. Mahaffey. 1988. "Lead and Osteoporosis: Mobilization of Lead from Bone in Postmenopausal Women." *Environmental Research* 47: 79-94.

Snee, R.D. 1981. "Evaluation of Studies of the Relationship Between Blood Lead and Air Lead." *Int. Arch. Occup. Environ. Health* 48: 219-242.

Taylor, T.N., P.H. Davis, J.C. Torner, J. Holmes, J.W. Meyer, and M. F. Jacobson. 1996. "Lifetime Cost of Stroke in the United States." *Stroke* 27(9): 1459-1466.

Tepper, L.B. and L.S. Levin. 1975. "A Survey of Air and Population Lead Levels in Selected American Communities." In: Griffin, T.B.; Knelson, J.H., eds. Lead. Stuttgart, West Germany: Georg Thieme Publishers; pp. 152-196. (Coulston, F.; Korte, f., eds. Environmental Quality and Safety: Supplement v. 2).

Tsuchiya, K., et al. 1975. "Study of Lead Concentrations in Atmosphere and Population in Japan." In: Griffin, T.B. and Knelson, J.H.. eds. Lead. Stuttgart, West Germany: Georg Thieme Publishers; pp.95-145. (Coulston, F.; Korte, F., eds/ Environmental Quality and Safety: Supplement v. 2)

U.S. Census. 1982. United States Summary, General Population Characteristics, Table 41: Single Years of Age by Race, Spanish Origin, and Sex: 1980.

U.S. Census. 1992. United States Summary, General Population Characteristics, Table 13: Single Years by Sex, Race, and Hispanic Origin: 1990.

U.S. Department of Commerce. 1976. Statistical Abstract of the United States: 95th Edition. Bureau of the Census. Washington, DC.

U.S. Department of Commerce. 1980. U.S. Census, United States Summary, General Population Characteristics.

U.S. Department of Commerce. 1986. Statistical Abstract of the United States: 105th Edition. Bureau of the Census. Washington, DC.

U.S. Department of Commerce. 1987. Census of Manufacturers.

U.S. Department of Commerce. 1990. *Earnings by Occupation and Education: 1990.* Bureau of the Census. Washington, DC.

U.S. Department of Commerce. 1990. U.S. Census, United States Summary, General Population Characteristics.

U.S. Department of Commerce. 1991. Statistical Abstract of the United States: 111th Edition. Bureau of the Census. Washington, DC.

U.S. Department of Commerce. 1992. Statistical Abstract of the United States: 112th Edition. Bureau of the Census. Washington, DC.

U.S. Department of Commerce. 1993. Money Income of Households, Families, and Persons in the United States: 1992. Bureau of the Census, Series P60-184.

U.S. Department of Commerce. 1993. Personal Communication between Bureau of Census, Population, Age and Sex Telephone Staff and Karl Kuellmer of Abt Associates on December 8, 1993.

U.S. Department of Commerce. 1994. *City and County Databook: 1994.* Bureau of the Census. Washington, DC.

U.S. Department of Commerce. 1994. Personal Communication between Bureau of Census, Population, Age and Sex Telephone Staff and Karl Kuellmer of Abt Associates on February 7, 1994.

U.S. Department of Education. 1993. Digest of Educational Statistics, 1993. National Center for Educaton Statistics, Office of Educational Research and Improvement. D.Ed. publication number NCES 93-292.

U.S. Department of Energy (DOE). 1990. Petroleum Supply Annual, 1989, Volume 1. DOE publication number EIA-0340(89)/1

U.S. Department of Energy (DOE). 1991. Petroleum Supply Annual, 1990, Volume 1. DOE publication number EIA-0340(90)/1

U.S. Department of Energy (DOE). 1992. Cost and Quality of Fuels for Electric Utility Plants 1991. DOE/EIA-0191(91) Energy Information Administration, August.

U.S. Environmental Protection Agency (U.S. EPA). 1984. *A Survey of the Literature Regarding the Relationship Between Measures of IQ and Income.* Prepared by ICF, Inc. Report to U.S. Environmental Protection Agency, Office of Policy Analysis, June.

U.S. Environmental Protection Agency (U.S. EPA). 1985. *Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis*. Prepared by U.S. Environmental Protection Agency, Office of Policy Analysis, Economic Analysis Division. February.

U.S. Environmental Protection Agency (U.S. EPA). 1986a. *Reducing Lead in Drinking Water: A Benefit Analysis*. Prepared by U.S. Environmental Protection Agency, Office of Policy Planning and Evaluation, Draft Final Report. December.

U.S. Environmental Protection Agency (U.S. EPA). 1986b. *Air Quality Criteria for Lead: Volume III.* Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA-600/8-83/028cF. June.

U.S. Environmental Protection Agency (U.S. EPA). 1987. *Methodology for Valuing Health Risks of Ambient Lead Exposure*. Prepared by Mathtech, Inc. for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Ambient Standards Branch, Contract No. 68-02-4323.

U.S. Environmental Protection Agency (U.S. EPA). 1990a. *AIRS Facility Subsystem Source Classification Codes and Emission Factor Listing for Criteria Air Pollutants*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-450/4-90-003. March.

U.S. Environmental Protection Agency (U.S. EPA). 1990b. *National Air Pollutant Emission Estimates 1940-1988.* Office of Air Quality Planning and Standards, Technical Support Division, National Air Data Branch. Research Triangle Park, NC. EPA-450/4-90-001.

U.S. Environmental Protection Agency (U.S. EPA). 1990c. *Review of the National Ambient Air Quality Standards for Lead: Assessment of Scientific and Technical Information.* OAQPS Staff Paper, Air Quality Management Division, Research Triangle Park, NC. December.

U.S. Environmental Protection Agency (U.S. EPA). 1991a. *National Air Quality and Emissions Trends Report, 1989.* Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA-450/4-91-003.

U.S. Environmental Protection Agency (U.S. EPA). 1991b. *The Interim Emissions Inventory.* Office of Air Quality Planning and Standards, Technical Support Division, Source Receptor Analysis Branch. Research Triangle Park, NC.

U.S. Environmental Protection Agency (U.S. EPA). 1992. *1990 Toxics Release Inventory.* Office of Pollution Prevention and Toxics, Washington, DC. EPA-700-S-92-002.

U.S. Environmental Protection Agency (U.S. EPA). 1994. *Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children.* February. EPA 540-R-93-081.

U.S. Environmental Protection Agency (U.S. EPA) database. Graphical Exposure Modeling System Database (GEMS).

Wallsten and Whitfield. 1986. *Assessing the Risks to Young Children of Three Effects Associated with Elevated Blood Lead Levels.* Argonne National Laboratory. December.

Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. "Medical Costs of Coronary Artery Disease in the United States," *The American Journal of Cardiology* 65: 432-440.